The Journal

OF THE

AMERICAN DENTAL ASSOCIATION

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ANATOMIC LANDMARKS OF VALUE IN FULL DENTURE CONSTRUCTION

By HAROLD LYTTON HARRIS,* B.A., D.D.S., St. Paul, Minn.

ANATOMIC landmarks of value in complete denture construction are recognized by the operator in direct ratio to his knowledge of the anatomy of the mouth and surrounding parts. It has long been my belief that a dentist should have a thorough knowledge of the normal size, shape, composition and function of the various anatomic structures underlying the mucous membrane of the mouth. That is his field and his responsibility. He should be as thoroughly familiar with its anatomic parts and their physiology as he usually is with the structure of a tooth. If he has this knowledge, he can more readily recognize the merit of the various impression technics that are before the profession today and apply them according to the requirements of each case rather than memorize or copy a series of successive steps.

Read before the Section on Full Denture Prosthesis at the Eighty-Second Annual Meeting of the American Dental Association, Cleveland, Ohio, September 11, 1940.

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The best impression technics obviously are those which produce dentures that when worn are in harmony with the structure and function of the tissues of the mouth. While there is a wide divergence of opinion as to how this can best be accomplished, there is also, as a direct result of the impression technic, a wide agreement on certain fundamental principles of full denture construction that must be built into the dentures. It is generally conceded that no one impression technic will ever be universally used by dentists. Dentists differ greatly in their ability to handle various impression materials and in their interpretation of the purposes for which the materials are used. Likewise, every mouth is different, and, many times, the problems presented, usually some variation in the anatomic form or function of the underlying tissues, cannot be solved by the particular technic that we are using. No technic used by the profession today can assure satisfactory results in every case if the dentist thinks only of the mechanical steps of the technic and fails to study the

foundation upon which he is attempting to build.

There are two fundamental principles which must be basic objectives of any good impression technic. 1. In order to take full advantage of the physical law of atmospheric pressure, in other words to produce a seal or suction, the borders of both upper and lower dentures must rest in soft tissue. 2. All dentures exert pressure on the underlying tissues under the force of mastication and this pressure must be distributed according to the resiliency of these tissues.

In regard to the first principle, placing the borders of a denture in soft tissue means, in the upper part of the mouth, that the border of the denture from the lateral side of one tuberosity around anteriorly to the lateral side of the opposite tuberosity must rest high in the buccal and labial cavities. As the border passes posteriorly around each tuberosity and from tuberosity to tuberosity across the palate, it must rest in resilient tissue and not on membrane of which bone is the most immediate underlying structure. In the lower part of the mouth, the same principle should be followed. The buccal, labial and lingual borders must be placed far enough from the crest of the ridge to come in contact with soft, resilient tissue at all points, and this also holds true where the border of the denture crosses the crest of the ridge in the posterior part of the mouth. It must be borne in mind that a denture, upper or lower, which rests only on mucous membrane of which bone is the most immediate underlying structure may have lateral stability, that is it may seat itself well in position, but it cannot have any resistance to the tipping stresses. This resistance is entirely dependent on the seal produced by placing the borders in soft tissue. In order that the borders may be in harmony with the structure and function of these tissues, it is necessary to take an impression in these areas in a material easily molded by the movements of the tissues, to produce what is generally known as a muscle-trimmed impression.

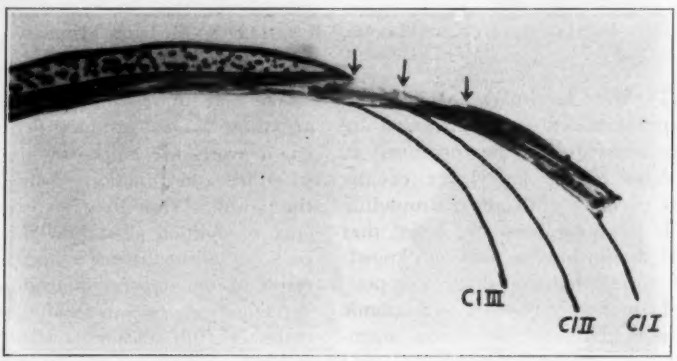

Fig. 1.—Schematic drawing showing relation of distal border of hard palate to movable tissues of soft palate, upon which House based his classification. Arrows indicate where tissues move when the patient says "Ah." In Class I and II types, the soft resilient tissue between the arrow and the distal edge of the hard palate is capable of supporting the postdam. In the Class III type, the tissue drops abruptly from the hard palate and the narrow postdam should be placed on movable tissue.

The second principle necessitates a study of the mouth to be fitted in order that the areas of different resiliency and the reasons for the variations in resiliency may be kept in mind. There is, of course,

a general rule or description for the average in these various areas. Blindly following such a rule or following a printed outline of the various steps in any given technic without first studying tissues to be included in the impression will lead to trouble in those cases in which there is some variation from the average pattern. Therefore, instead of attempting to apply a given technic by simply going ahead and following a series of steps, it is better to examine the mouth first to determine whether the various anatomic landmarks require any modification in the impression steps. This preliminary examination will prove to be time well spent. It will give the operator a better mental picture of what he is trying to accomplish and warn him when modification is necessary.

In the examination preliminary to taking impressions for complete dentures, let us consider the upper part of the mouth first. While general conditions should, of course, be noted, the most important part to examine is that area in which the posterior line of the denture and the postdam will be placed. I determine first of all the classification of the throat form. (Fig. 1.) This procedure is based upon the teaching of House and consists of three classifications. Class I presents at least 5 mm. or more of resilient tissue which does not move when the patient says "ah" and which is capable of withstanding the postdam pressure, between the posterior border of the hard palate and the posterior plate line. Class II includes those cases having from 1 to 5 mm. of this type of tissue, and Class III those cases in which the posterior border of the hard palate on the median line is at the junction of the movable tissue of the soft palate.

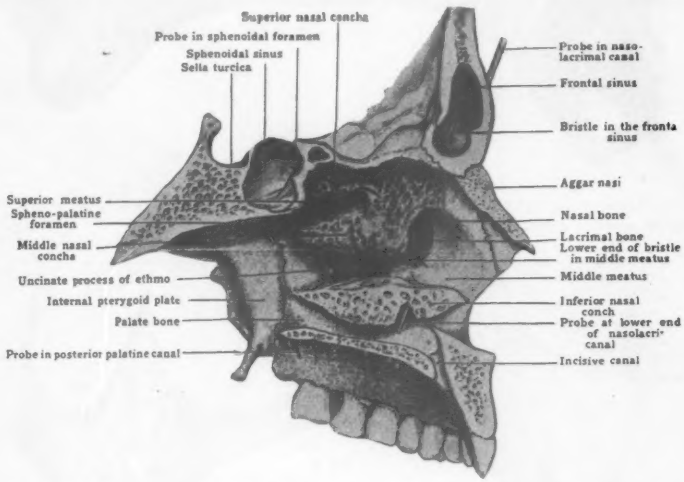

Fig. 2.—Medial view of maxilla (Morris) to show relation of hamular process of sphenoid bone to tuberosity. This notch may be found easily by moving the mouth mirror posteriorly to the tuberosity.

In order to determine the classification of any mouth, the posterior denture line should first be determined. Many operators freely admit that they have no definite way of knowing where this denture border should be. Yet when one realizes the requirements of the posterior

border of the upper denture, there is neither doubt nor guesswork, according to the anatomic structures in that area, where the denture line should be placed. The border along the posterior denture line is the only border of the upper denture which is not formed by the functional movements of the underlying tissue. It is arbitrarily placed and developed by manipulation. Since postdamming can be interpreted only as an ever possible, just anteriorly from the point where the tissue of the soft palate begins to move when the patient says "ah." Ordinarily, the two little dots that appear on each side of the median line in this area can be taken as a landmark for the posterior denture line, but this must be determined in the mouth and not guessed at on a cast. An exception to this statement which occurs in Class III will be explained later.

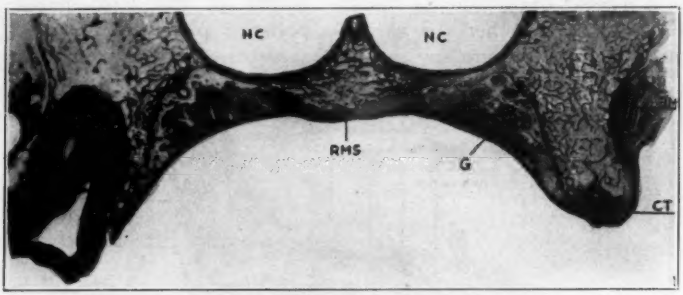

Fig. 3.—Cross-section through maxilla (Pendleton) in third molar area, showing comparative thickness of mucous membrane across palate which will support denture.

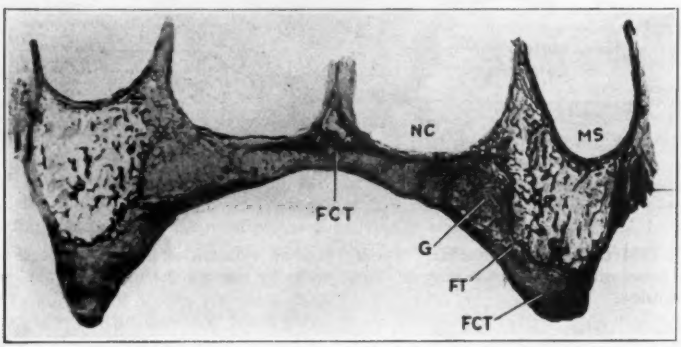

Fig. 4.—Cross-section of maxilla (Pendleton) through third molar area, showing, as in Figure 3, thickness of mucous membrane across palate. The architecture of the bone, always producing the thickest portion of the mucous membrane in the region of the posterior palatine canal, is to be noted.

increased compression of the underlying tissues along the posterior border of the denture, that pressure must be exerted on tissues capable of withstanding the pressure. In the median line, the posterior denture line should be placed, when-

In the area of the tuberosity, either the right or the left side, there is only one location which permits the underlying tissue to carry a postdam pressure, and that is the space between the tuberosity and the hamular process of

the sphenoid bone. (Fig. 2.) This notch is easily found in any mouth by placing the edge of a mouth mirror on the crest of the ridge in the molar area and moving it distally beyond the tuberosity until it drops readily into the triangular space created by these two osseous structures. The underlying tissue in this area is a type perfectly capable of withstanding the increased compression of the posterior border of the denture. The denture border, if too short in this area, rests on mucous membrane overlying bone with insufficient resiliency to maintain the postdam long. If the denture is extended distally so that the postdam pressure is exerted against the hamular process, we not only encounter less resiliency of tissue, but also interfere with the functional movements of the tensor veli palatini muscle, and the patient experiences a sharp pain in the act of deglutition, or swallowing. This non-resiliency of the overlying mucous membrane. Unfortunately, too many dentists, and this fact is borne out by figures from dental laboratories, leave

notch between the hamular process and the tuberosity should be carefully examined on both sides so that we can visualize the exact path of the posterior denture line across the mouth.

The second area to be examined in order to arrive at the classification is the hard palate. Its characteristics in regard to size, shape, extent and apparent thickness of overlying mucous membrane must be determined. Pendleton has demonstrated by a series of cross-sections of the maxillae (Figs. 3 and 4) that the hard palate varies considerably in these characteristics. It is generally conceded as a fundamental step that the hard palate must be relieved because of the

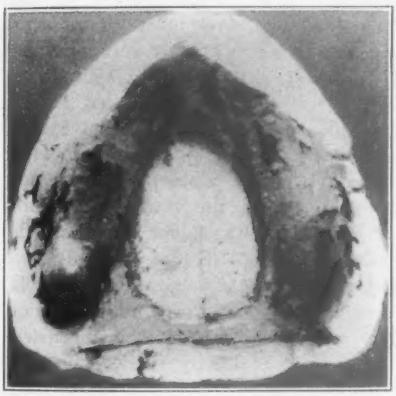

Fig. 5.—Upper impression for complete denture, showing tracing of hard palate and posterior denture line transferred to impression. The outline of the area to be relieved can now be transferred to the cast by making a fine line in the outline of the impression, which will appear as an embossed line on the cast; or relief can be obtained by scraping the impression within the outline. The posterior denture line is also grooved for transfer to the cast.

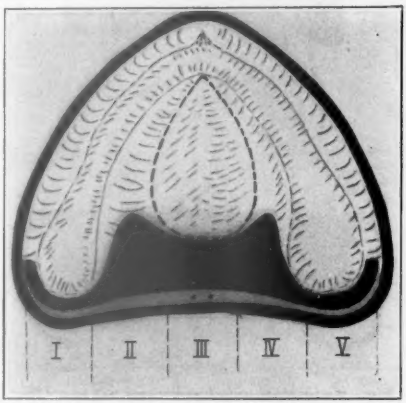

Fig. 6.—Diagrammatic view of posterior technic in Class I case. Postdam in areas I and II is the width of a No. 10 round bur, and also the width of the first cut made across all five areas. The width of the postdam in area III is definitely from the posterior denture line beginning of the relief area. In areas II and IV, the postdam is widened as indicated, the compression anteriorly diminishing as less resilient tissue is encountered. In a Class III type of throat form, the width of the postdam in area III would be the same as in areas I and V.

the matter of relief to the dental technician, who is supposed to determine on an inanimate plaster cast just what areas need relief. The majority of cases are sent to the laboratory with the words, "Put in relief," without any further word or indication as to how much or where it is to be placed. The average commercial laboratory, in order to save time, has its tinfoil cut into three graduated sizes, ready to be placed in the center of the cast.

If there is any step in full upper denture construction that should be taken at the chair, it is that which determines the exact area to be relieved and the amount of relief required to counteract the settling of the denture. It takes but a few minutes with a ball burnisher or any other small, blunt instrument to study the resiliency of the tissue of the hard palate and to determine where the non-resilient tissue ends and the resilient tissue begins. This will indicate the outline of the hard area and later should be charted on the membrane with an indelible pencil, to be transferred to the impression as it is completed. (Fig. 5.) If we are not making this particular step, we shall find that, in most cases, the relief must go farther posteriorly on the median line than any commercial laboratory will ever extend it. We find also that many cases need more relief on one side of the median line than on the other, and this is never accomplished by placing three pieces of tinfoil in the center of a plaster cast. Anteriorly, the relief should always be made over the anterior palatine foramen to avoid compression of the tissue overlying the blood vessels and nerves that emanate from this foramen.

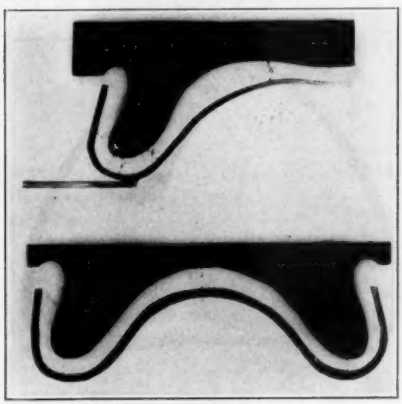

Fig. 7.—Relation of tray to ridge. The tray must be large enough to assure at least one-quarter inch thickness of compound at all points, and long enough anteroposteriorly to include tissues beyond the posterior denture line.

Now that the posterior border of the hard palate has been charted on the membrane as well as the posterior denture line, we can readily recognize the classification of throat form with which we are working. Classes I and II are favorable cases because they have resilient tissue capable of withstanding the postdam pressure between the posterior border line and the posterior denture line. Class III, in which the posterior border of the hard palate is found to go back to the movable tissue of the soft palate, presents an unfavorable type of case. This is known as the shortened throat form, and in these cases the operator has two choices. If he elects to place his posterior denture line anteriorly to the movable tissue of the soft palate, he must place it without a postdam in the median line, inasmuch as the underlying tissue is non-resilient. The result is a denture that will not resist the tipping force produced by leverage on the anterior teeth. The other choice, and the lesser of the two evils, is to place the posterior denture line in the movable tissue of the soft palate. The unfavorable part of this procedure is that it very often produces a tickling or gagging sensation from the movements of the soft palate. However, I have found that when I explain to the patient the differ-

ence between the two areas and secure his cooperation, tickling or gagging can be overcome in a week or two, and the patient has a much better denture as a result. In this type of mouth, the postdam, while it is kept narrow, is placed over tissue capable of withstanding the pressure. Therefore, as can be readily seen, determining the posterior plate line is likewise a step to be made by the operator at the chair and not to be left to a technician at a bench in some far-away laboratory.

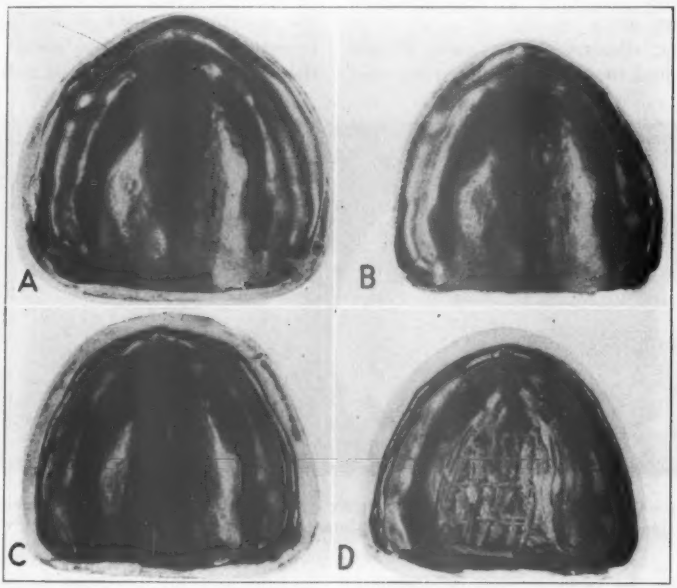

Fig. 8.—Four steps in upper impression technic: A, first insertion for distribution of material, to include all desirable areas. B, excess on labial and buccal trimmed back to height of convexity of compound in labial and buccal cavities, preparatory to starting the muscle trim. The compound tray is not trimmed up to the posterior denture line, but is left at least one-fourth inch too long. C, compound completely muscle trimmed, which indicates height and thickness of denture borders in labial and buccal cavities. D, compound tray prepared for finishing material.

The area to be postdammed along the posterior denture line should also be examined in order that the operator shall have in mind the various degrees of resiliency of the tissues in this area. Again, a general description fits the average case, but because there is no way of determining that the patient in the chair presents an average case, it is important to examine these areas. This should be carefully done whether the operator intends to accomplish the postdamming in the impression or on a cast. If the impression technic calls for postdamming the case on the cast, the operator would do well to make this examination at the second visit.

However, let us consider this area in detail. For convenience, we divide the posterior denture line into five equal areas, I, II, III, IV and V. (Fig. 6.) Areas I and V are not necessarily identical, but are similar, consisting of the space between the tuberosity and the hamular process. While the latter is

considered a constant structure in anatomy, the size and shape of the tuberosities vary greatly. On one side, we may have a full, rounded tuberosity; on the other side, a condition in which no tuberosity whatsoever seems to be present. Perhaps the past history of the third molar and the manner in which it was removed have a bearing on this condition. Nevertheless, the notch or triangular space can always be located. This is found to be a space that averages about one-eighth inch in width. Therefore, we must plan the postdam in these two areas so that it is no wider than that dimension. In areas II and IV, we find the most resilient tissue and, therefore, the postdam can be widest at this point. This is due to the structure of these areas (Figs. 3 and 4); that is, the osteologic structure of the palate and the nature of the associated soft tissues. The bone of the alveolar process is formed at a right angle to the palate bone, while the mucous membrane curves over the palate, producing in areas II and IV the greatest thickness of mucous membrane in the roof of the mouth.

In area III, which is the median line area, the width of the postdam is determined by the posterior border of the hard palate and the posterior denture line itself. The depth of postdam that can be attained is determined by the nature of the anatomic structures in these five areas. In areas I and V (Fig. 9), the space between the tuberosity and the hamular process contains nothing more than connective tissue, or fascia, perfectly capable of withstanding compression, and it is only in overextension in this area that the border of the denture bears against the bursa of the tensor veli palatini muscle.

In areas II and IV, we have not only the greatest amount of mucous membrane in all of the five areas, but also the insertion of the tensor veli palatini muscle into the posterior border of the palatine bone. Because the postdam here is placed on mucous membrane plus muscle fiber, we get the greatest depth possible. In the median line area, or area III, we have as underlying tissue the aponeurosis of the levator veli palatini muscle. The term aponeurosis signifies the point of insertion on the median line of like muscles of opposite sides and is a thickened form of the connective tissue, or fascia, which means that it is less resilient, and we find in the average mouth that area III has the least resi-

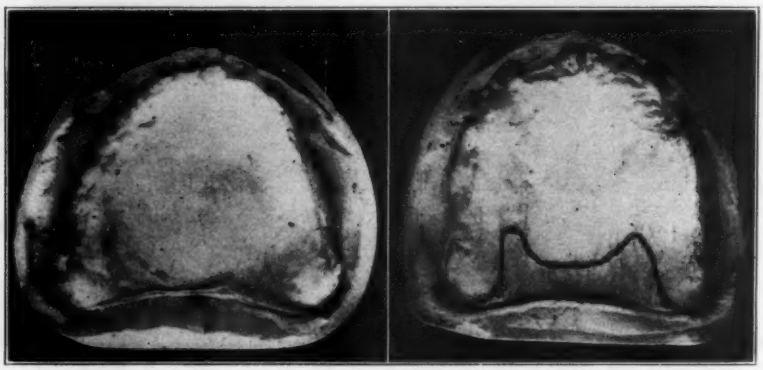

Fig. 9.—Casts from impression as in Figure 5, showing embossed line indicating posterior denture line, and outline of areas to be postdammed, not by guesswork, but by study of mouth.

lient tissue, and, therefore, the least postdam pressure is possible.

How much postdam pressure should there be? How is the amount determined? By what method can it best be incorporated in the finished dentures? These are interesting questions, and many opinions have been stated and many methods advocated in answer to them. Perhaps the easiest, quickest and least accurate answer is to leave it to the laboratory. Thus no valuable time is lost, and there is no holding up of patients in chairs two and three. Simply include in the instructions something like this. "After you have pasted in the tinfoil relief, figure out where the posterior denture line should be and put in a little postdam for me." This can even be dictated to an assistant while the impression for the next patient is being started. Another method, having much more possibility of accuracy and practiced by many good operators, is to build up the postdam on the impression by the addition of some material which will compress tissue. It is a method which requires expert manipulation and is fraught with hazards of error. If the postdamming material is too cool, the tissue will be overcompressed; if it is too warm, there is more flow than compression, and the flow is difficult to control. In other words, it is difficult for the operator to control the width and depth of the postdam.

There is a method of building the postdam, however, which is not difficult to handle and which has a far greater possibility of accuracy, because the operator is in complete control of the situation, and the final result as to width and depth is entirely the result of his judgment, based upon careful examination of the tissues to be compressed. The following technic is suggested:

A tray is selected for the upper impression which is long enough anteroposteriorly to insure an impression of the tissues approximately one-fourth inch distally from the posterior denture line. If necessary, the tray edges are bent down along the posterior edge to allow escape of compound and to prevent impingement of the metal edge against the mucous membrane. A compound im-

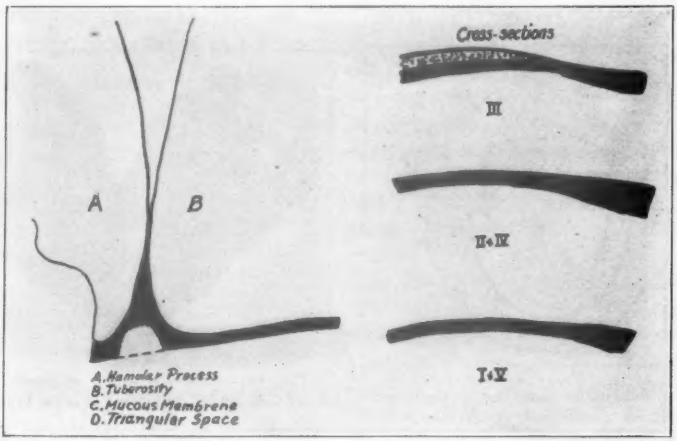

Fig. 10.—Schematic drawing of depth and width of postdam in various areas as indicated in Figure 6.

pression is taken, trimmed of excess and removed from the metal tray after it is certain that the palatal portion of the compound has no less than one-fourth inch thickness at all points. (Fig. 7.) The compound impression is next muscle-trimmed carefully (Fig. 8 a, b, c) and then is prepared to be used as a tray for the finishing material by reducing or cutting away no less than one-sixteenth inch and no more than one-eighth inch of compound in the area to be relieved and across the posterior denture line from area I to area V. This results in a compound impression tray that compresses tissue when pressed to place only over the stress-bearing ridges.

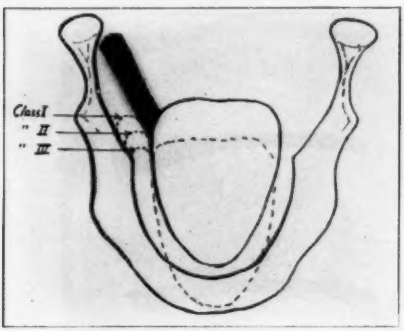

Fig. 11.—Schematic drawing to show relation of mucous membrane overlying palatoglossus muscle, when tongue is protruded to point just below red line of lower lip, and resulting size of retromolar fossa to determine lateral throat form classification.

(Fig. 8 D.) Any finishing material, such as a good impression plaster (Fig. 5) in a thin, creamy mix, or any of the new impression materials that will not displace soft tissue when mixed ready for insertion in the mouth, is satisfactory. This results in an impression of the hard palate to be relieved and the tissues to be postdammed under no compression whatsoever, and the cast poured in this impression represents these tissues in their rest position. At this stage, the posterior plate line and the hard palate are traced on the mucous membrane with an indelible pencil and transferred to the impression either before or after the finishing material is used.

The relief required is accomplished by scraping the impression, or this can be deferred until the cast is poured. If the latter, a fine groove is made with an explorer in the outline of the hard palate, which will appear on the cast as a fine embossed line, eliminating any guesswork as to where the relief should be.

A groove is likewise made along the posterior denture line on the impression. This also appears as an embossed line on the cast (Fig. 9), eliminating any

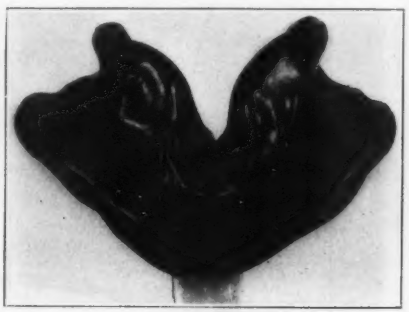

Fig. 12.—Lower primary impression, which must include all available seating area of denture. Digital pressure must aid in pressing the compound into the distolingual or retromolar fossa.

guesswork or indecision as to where this important border should be.

After the cast is separated, a strong trial denture is constructed, and, with the patient in the chair, the procedure of postdamming is started. Using a suitable instrument, such as a ball burnisher or a mouth mirror, the operator compresses the tissue in area I to the fullest extent, estimating in terms of millimeters the amount of compression, or the upward path of the instrument from the point where it just touched the surface of the membrane until complete

compression is reached. Two-thirds of this distance is then computed, and, with a No. 10 round bur in the straight handpiece, a groove that depth is cut into the cast along the embossed line in area I. For example, if the amount of complete compression is found to be 3 mm., a groove 2 mm. in depth is cut into the cast. Two-thirds of the amount representing complete compression is used so that the finished denture will not completely compress the underlying tissue and thus cut off the blood supply and nutrition. The No. 10 round bur is used because it is the width of the space available for the postdam between the hamular process and the tuberosity.

The operator then studies area V and

Fig. 13.—Cast from lower primary impression outlined for construction of tray for taking master impression. The outline includes the external oblique ridge on the distobuccal aspect, the retromolar triangle on the crest of the ridge and the retromolar fossa on the distolingual aspect.

applies the same technic. Next, he prepares the groove in area III, the median line area, in which the smallest depth of compression is usually allowable, but whatever is determined by a study of the tissue in this area is transferred to the cast by cutting the groove the proper depth. Then the groove through area I is connected with the groove in area III by cutting into the cast in area II the proper amount as determined by examination. Area III is then similarly connected with area V through area IV. We then have a groove the width of a No. 10 bur across the five areas of the posterior plate line as indicated (Fig. 6), which varies in depth according to the resiliency of the underlying tissue, but does not take into consideration the width of the postdam that may be placed. Therefore, with a sharp knife, we widen the postdam in area III from the depth of the groove to the posterior border of the relieved hard area, reducing the pressure anteriorly so that, at the anterior borders of the postdam pressure, the plaster is cut to a featheredge. (Fig. 10.)

In areas II and IV, we find that we can apply the greatest width of postdam pressure, which should run anteriorly a depth of from 10 to 15 mm. Many operators urge that, instead of applying a postdam pressure to this particular area, relief is necessary because of the presence of the posterior palatine canal. The anatomic structure of this area as found in dissection has shown that the mucous membrane overlying this area reaches an average depth of from 15 to 20 mm. and that the blood vessels and nerves which emanate from this foramen run anteriorly in a bony groove which protects them for more than half their thickness; therefore this area can stand postdam pressure rather than be relieved, and a more comfortable denture results.

In areas I and V, the width of the postdam must be confined to the width of the No. 10 bur; therefore, only that part of the cast is scraped away to reduce the sharp anterior edge of the groove. If the postdam pressure is widened anteriorly in this area, pressure is exerted upon the thin mucous membrane overlying the posterior part of the tuberosity. We know that cellular atrophy results from this type of pressure; therefore, a shrinkage of tissue takes place, causing a leakage in that

area of the denture. If the postdam is widened posteriorly so that pressure is brought to bear against the hamular process, a condition known as bursitis results because of the inflammation caused on the bursa of the tensor veli palatini muscle as it turns around the hamular process. With this condition present, the patient undergoes sharp pains in the act of deglutition, and the denture would be expected to resist. If there is air leakage anywhere across the posterior denture line, it is readily seen and can be corrected by deepening the postdam on the corresponding area of the cast. While shortage of a postdam can be detected, overcompression cannot be, but it is well to remember that to overestimate the amount of compression that any area can tolerate is bet-

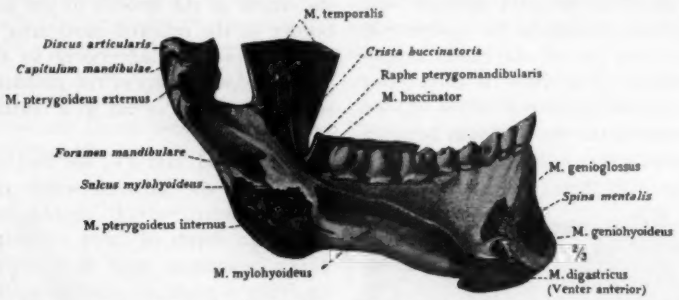

Fig. 14.—Lingual view of mandible (Morris). The terminal fibers of the temporal muscle in the area of the retromolar triangle and the relation to the buccinator fibers are to be noted.

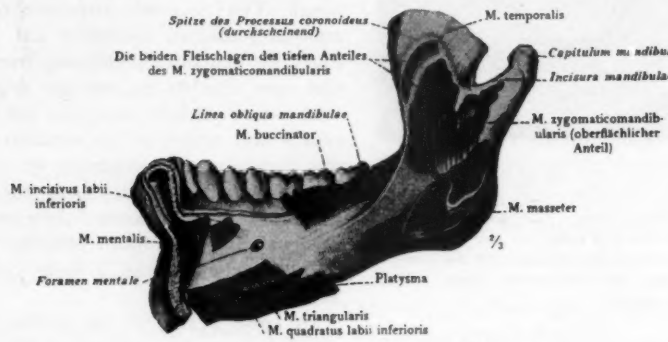

Fig. 15.—Lateral view of mandible (Morris).

denture must necessarily be shortened at that point.

After both depth and width of postdam have been established on the cast, the posterior portion of the trial plate is heated, the trial denture is reseated on the cast and the warmed portion is pressed and adapted to the postdam. It is then tried in the mouth and given the same tests of stress that the finished denture would be expected to resist. If there is air leakage anywhere across the posterior denture line, it is readily seen and can be corrected by deepening the postdam on the corresponding area of the cast. While shortage of a postdam can be detected, overcompression cannot be, but it is well to remember that to overestimate the amount of compression that any area can tolerate is better than to underestimate it, because, if any spot is overcompressed by the finished denture, the postdam at that point can be reduced by polishing; whereas, if the finished denture lacks sufficient postdamming, it is quite difficult to increase it.

Another condition that may modify the impression technic used by the operator is that of the mucous membrane

on the crest of the ridge. If the membrane is thick and flabby, he must make whatever modification is necessary in his technic to take an impression of this tissue in its natural position. In other words, he must be careful in his choice of materials or in their manipulation that no pressure is created to place these tissues in an unnatural position. If one is using compound, a window or groove can be cut into the impression to allow this soft, flabby tissue to remain in its natural position when the balance of the impression is compressed against the other tissues of the mouth. Some sort of low-fusing material or a thin mix of plaster must be used to finish this type of impression.

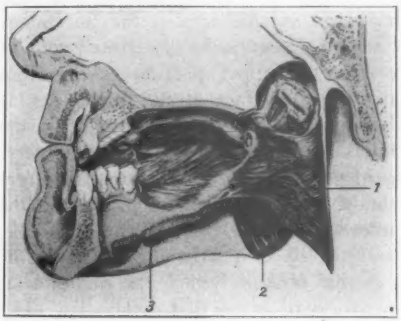

Fig. 16.—Lingual view of mandible and structures forming buccal cavity. The relation of the mylopharyngeal muscle (lower portion of superior constrictor) to the mylohyoid muscle in the retromolar fossa is to be noted.

The lower part of the mouth presents many more anatomic landmarks than the upper. Here, too, my first concern in examining the patient prior to taking the impression is to determine the lateral throat form classification as advocated by Neil. Here, the three classifications are based upon the size of the retromolar fossa when the tongue is in a given position. (Fig. 11.) With a mouth mirror or the little finger in the retromolar fossa and the patient protruding the tongue just beyond the red line of the lower lip, the size of the retromolar fossa is determined by the distance between the mylohyoid line and the surface of the mucous membrane as it is pulled forward. When the tongue moves forward to this position, it carries with it the palatoglossus muscle and the overlying mucous membrane. In Class I, there remains as the retromolar fossa a circular space about one-half inch or more in diameter. In a Class II throat form, this space is smaller; and, in a Class III, the least favorable type, the mucous membrane overlying the palatoglossus muscle is pulled directly up to the mylohyoid line, and there is no retromolar fossa.

The importance of this classification is in the information it gives the opera-

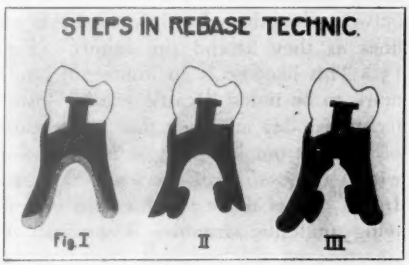

Fig. 17.—Technic to "lift" soft tissue of lower ridge into natural position. I, light area indicating space for impression material, preferably compound. II, application of impression material on sloping sides of ridge. III, after first insertion and incomplete closure at crest of ridge, additional impression added in same location as in II.

tor as to the extent of the tissue posterior to the mylohyoid line as it curves upward to the ramus, which the denture may cover to insure the border's resting in soft tissue and still be in harmony with the functional ranges of these tissues. Thus, if the operator is working with a Class I case and is using a lower impression technic in which he must take a preliminary impression in order to construct a properly fitting tray, he must make certain that his impression

material reaches well into the retromolar fossa. (Fig. 12.) Later, he is careful to trim the tray for the master impression to the proper size to be tolerated in this fossa. If he is working with a Class III case, he knows the distolingual border must reach distally just beyond the mylohyoid line, similarly to placing the posterior denture line of the upper denture in a Class III case, even though it infringes upon the functional range of the soft tissue. If the border is overextended, soreness results; and if it is too short, the operator has violated the first principle.

Perhaps it would be well here to distinguish between the retromolar triangle and the retromolar fossa. The retromolar triangle is the pear-shaped pad of tissue on the crest of the ridge which lies between the internal and external oblique lines as they ascend the ramus. (Fig. 13.) This likewise is an important landmark to be noted because leading lower technics today advocate that all or most of this retromolar triangle be included within the outline of the lower denture. It is a pad of tissue in which the underlying anatomic structure is the area of insertion of the terminal fibers of the temporal muscle as they come down from the temporal fossa to be attached to the coronoid process and along the anterior face of the ramus as far as the crest of the alveolar ridge. (Fig. 14.) The retromolar fossa is that area on the lingual portion of the mandible distally from the mylohyoid line at its most prominent point, and its size is determined by the functional movement of the palatoglossus muscle and overlying mucous membrane upon protrusion of the tongue. This muscle, as you know, has its origin in its aponeurosis on the median line in the soft palate, passes laterally and downward, forming the anterior pillar of the tonsillar crypt, then curves medially to be attached to the lateral surface of the tongue. Many times, it is a heavy, cordlike muscle and, in such cases, it can be as difficult to control as is the mylohyoid muscle of the floor of the mouth where retention of the lower denture is concerned.

Another important anatomic landmark is the external oblique line. (Fig. 15.) One of the most popular lower technics before the profession today advocates placing the borders of the denture arbitrarily over the external oblique line. Whether or not one believes in this technic, it is fundamental that the denture border be placed as far buccally as the functional range of the border tissue will permit, even encroaching upon the functional range of the border tissue to insure the denture border's resting in soft tissue. The underlying anatomic structure in this area is the buccinator muscle. This muscle has three points of origin, the upper portion arising from the lateral surface of the maxilla as far forward as the first molar area; the middle portion arising from the pterygoid-mandibular raphe, which runs from the tip of the hamular process to the internal oblique line of the mandible, and the lower portion arising from the external oblique line of the mandible as far forward as the first molar area. (Fig. 16.) The fibers converge, to become attached in the orbicularis oris at the corner of the mouth, determine the lateral wall of the oral cavity and serve as an auxiliary muscle of mastication.

The mucous membrane overlying the alveolar ridge should likewise be examined. If it is loose and flabby, the technic must be modified, if necessary, so that the impression will be taken with this tissue in its normal position and under no compression. This, which is described as "lifting the tissue" in its normal position, can be accomplished, for example, by a technic which calls for the application of the impression material along the sloping sides of the alveolar ridge, and as the material is

pressed to place, the flowing compound or wax tends to meet the material from the opposite side at the crest of the ridge, thereby lifting the tissue into its normal position. (Fig. 17.)

These points, if kept in mind, and the ability to modify the technic used by the operator in order that these important features may be included in the construction of the dentures, will save much time after the case is completed and will result in a more satisfactory case. Variations in form and size of these underlying structures are recognized only if the operator is thoroughly familiar with the structures in his field. As the Army Intelligence furnishes an Army commander with advance information concerning the enemy in order that he may plan an attack to take advantage of this information, so a preliminary examination of the patient's mouth before taking the impression gives us information to help in planning our procedure. To be forewarned is to be forearmed, and the little time that it takes to make this preliminary examination will repay the operator many times by adding to the success of his dentures.

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SOME BENIGN SURFACE LESIONS OF THE ORAL REGION

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A REASONABLE understanding of the pathologic tissue reactions in any manifestation of disease is important regardless of whether the operator is to attempt treatment or is to refer the patient to the specialist. In the oral cavity, a multitude of surface lesions are seen, many of which have a similar clinical appearance, but are histologically quite different. Frequently, it may be necessary to separate benign from malignant entities before a plan of treatment can be considered. This differentiation is possible only with a knowledge of the specific tissue changes peculiar to that lesion. The biopsy is an excellent means of assisting in this decision, provided a sizable specimen from a representative area, together with a complete history, is submitted.

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It is not the purpose of this paper to present either the historical background or the clinical picture of these reactions except as such information is indicated in discussing the histologic picture. It is believed, however, that there is some indication for a comparative analysis of the microscopic features encountered by the pathologist. With this thought, therefore, the tissue reactions in some of the interesting and frequently more complex oral surface lesions will be discussed.

LEUKOPLAKIA

One of the most common tissue reactions encountered in and about the oral cavity is leukoplakia. To present this lesion as either a benign or a malignant manifestation will evoke certain differences of opinion. A great many of the observations on this subject have been made by authors studying leukoplakic vulvitis. If definite diagnostic criteria

are established, leukoplakia, in either location, is essentially the same.

Novak[1] states that leukoplakic vulvitis, "perhaps more than any other gynecological lesion, exerts a strong tendency to the development of cancer." Taussig,[2] after considerable study of leukoplakic vulvitis, says, "I believe [this lesion] will inevitably proceed to the development of a cancer, but that the rate at which this change takes place may be so slow that death supervenes before accomplishment." MacLeod and Muende[3] believe that leukoplakia of either the mouth or the genitalia may occasionally develop into cancer, but that "this is a rare happening and only occurs when the leukoplakia is especially thick, superficially eroded and fissured, and possibly as a result of unsuccessful irritating treatment." Regardless of this diversity of opinion, it is fairly well established that malignant changes may occur in these lesions.

Taussig[2] divided the course of the disease into a hypertrophic and an atrophic stage, the dividing line between which it is generally difficult to determine. Keratotic activity, either a hyperkeratosis or parakeratosis, or both, with accompanying marked acanthosis and a varying degree of inflammatory exudate, is characteristic. The former is responsible for the tough leathery surface. The inflammatory elements are, in most instances, confined to the upper portion of the corium and are accompanied by frequent edema and fibrosis. These reactions will vary in degree not only in different lesions, but also in the same section. The granular layer of the epithelium may be more prominent than usual and some degree of dyskeratosis may be present. The acanthosis not infrequently causes a broadening and fusing of the rete pegs. Hydropic degeneration of the prickle cell layer may be seen. (Fig. 1.)

Whether all of these features are essential to the diagnosis of leukoplakia is questionable. However, most of these changes are seen in advanced cases. It would seem that the application of the term "precancerous" to obviously benign lesions is questionable unless one thinks of the possibility of this term being defined as a lesion in whose wake may follow another separate and distinct lesion of definite malignancy. From the standpoint of definitive treatment, all lesions must be considered as either benign or malignant. It would follow, therefore, that borderline malignancy should perhaps be reported conservatively lest

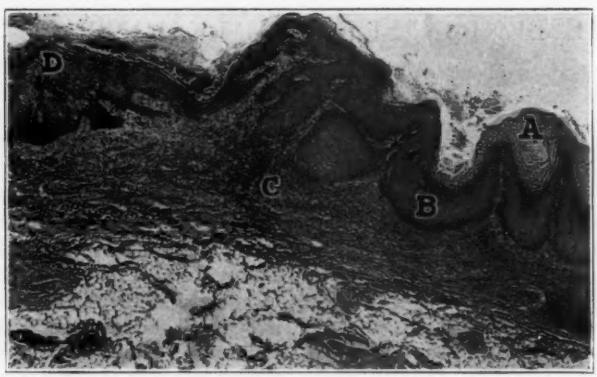

Fig. 1.—Leukoplakia. (×36.)

radical treatment follow where not specifically indicated.

BASAL CELL CARCINOMA

Basal cell carcinoma is frequently seen in the region of the cheek, nose or eyelid and occasionally on the lip. Its presence on mucous membrane is quite rare. Bell[4] states that "some tumors of the mucous membranes bear a resemblance to basal cell carcinoma of the skin, but they are much more malignant." Most authors consider this tumor relatively benign, the possibility of metastases being very slight. The term "rodent ulcer" has been applied because of the tendency to erosion of the deeper tissues, resulting in loss of considerable structure.

Much diversity of opinion exists as to their origin. Haythorn[5] believes that they arise from the sebaceous glands and the hair follicle matrix. Boyd[6] states that there are "three possible sites of origin—the basal cells of the epidermis, the hair follicles and misplaced cell groups from the epidermis," but that their being frequently situated in the line of embryonal fissures on the face supports an origin from misplaced cell groups.

Microscopically, numerous cell strands or clumps are present in the submucosa, usually extending to a constant level and having rounded or clubbed ends. These cells have basal characteristics, being spindle shaped, staining very dark and having a definite nuclear membrane and prominent nucleoli. No prickles are seen. Mitotic figures are usually absent. However, in rapidly growing tumors, they may be seen occasionally. Necrosis and ulceration are frequently seen with little tumor tissue remaining. The absence of keratinization or pearl formation is accounted for, according to Krompecher, by the fact that these tumors arise from the lowest layer of basal cells. In spite of their slow growth and slight tendency to metastasize, he tends to associate them closely with the nevi from which the very malignant melanomata may arise. This association, according to MacCallum,[7] seems questionable. These tumors are unusual in that they are quite "undifferentiated," and yet are relatively benign and respond well to irradiation. (Fig. 2.)

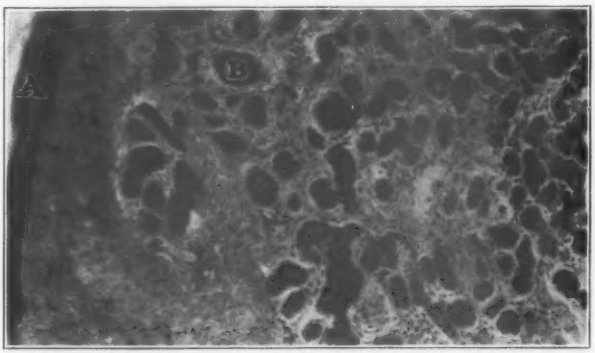

Fig. 2.—Basal cell carcimona. (×165.)

NEVUS

From a dermatologic standpoint, the term "nevus" is generally applied to most of the congenital circumscribed anomalies of the skin. Generally speaking, however, the term is more accurately employed in referring to the "pigmented mole." Marked vascularity may be seen in some instances and in others hypertrophy and hyperplasia of the hair

follicles. It may be seen, then, that these lesions present a variety of pictures, in some instances resembling superficial angiomata. An unusual degree of variance in opinion as to their origin has existed in the past. They have been described, at one time or another, as arising from all three of the embryonic layers. The presence of chromatophores has tended to support the theory of mesoblastic origin. However, most agreement is met in the theory of Masson, who considers their origin neurogenic.

Microscopically, they consist of collections of clear cells grouped in a characteristic manner in the upper portion of the corium. The cells are usually packed close together, the melanoblasts, if present, being situated at the outer edge of these clusters. Masson's trichrome stain demonstrates the presence of medullated and non-medullated nerve fibrils throughout these cell collections. The amount of pigment will vary considerably, although it is generally considered that this has no relation to the possibility of malignancy. Malignant changes are not impossible, however, particularly in the cutaneous forms, resulting in the malignant melanoma, a highly dangerous lesion. The hypertrophy of the hair follicles or the sebaceous glands may be an important feature, sufficient, in the minds of some authors, for the diagnosis of nevus, even in the absence of nevus cells.

The epidermis may be atrophic or may exhibit varying degrees of acanthosis with occasional hyperkeratosis and a rugose surface. In many cases, the nevus clusters appear to be separated from the epidermis by a clear zone of connective tissue, interpreted by some as an indication that these cells may have become detached from the epidermis during embryonic life. (MacLeod and Muende.[3]) The presence of these lesions on oral mucous membrane is rare. I have seen only one such case. The lesion, about the size of a dime, occurred on the buccal mucous membrane over the upper left central and lateral incisors in a white woman aged 27. It was non-pigmented, rugose and of long standing. (Fig. 3.)

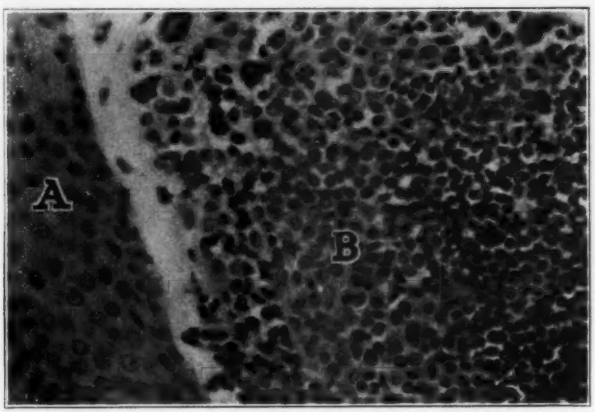

Fig. 3.—Nevus of buccal mucosa. (×315.)

LICHEN PLANUS

The importance of this disease lies in its differentiation from leukoplakia. It is not considered an important lesion since subjective symptoms are very slight, if not absent entirely. The prognosis is favorable, the course differing from leukoplakia in that the eruptions usually disappear spontaneously, whereas leuko-

plakia tends to persist. In ten cases, Fox[8] found that the average age of occurrence was 44.6 years, and the duration at the time of the report averaged four years. All of these cases involved the mucosa of the cheeks and six also involved the tongue. Chronic irritation appears to have an important rôle in the etiology, along with certain systemic dis-

Fig. 4.—Lichen planus. (×165.)

eases, such as syphilis and gastrointestinal disturbances. Little, however, is definitely known as to the cause of numerous other cases in which these factors could not be demonstrated.

Generally speaking, these lesions are characterized by an acanthosis of the superficial epithelium with a hyperplasia of the prickle cell layer. This frequently leads to a broadening of the rete pegs. The keratotic activity, as in leukoplakia, may manifest itself as either hyperkeratosis or parakeratosis. These changes in the epidermis, together with hydropic degeneration of the prickle cells, give the white shiny angular appearance characteristic of the lesion. In the immediate corium, a dense chronic inflammatory reaction is usually present, extending to the basal layer and in many areas actually invading it. This nearness of the inflammation is characteristic, as is the sharp demarcation of the inflamed area from the deeper portions of the submucosa. Frequently, a diagnostic fea-

ture is the presence of cystlike areas between the basal layer and the immediate corium. These areas at first may appear to be artefacts, but, on closer scrutiny, are seen to be areas of cystic degeneration of the basal cells. Endothelial proliferation is consistent with that seen in other inflammatory reactions. (Fig. 4.)

Malignant changes or the subsequent appearance of cancer in these eruptions are not common, although such cases have been reported. Saad[9] reports the appearance of a squamous cell epithelioma in a lesion on the buccal surface of the cheek of three years' duration, the diagnosis having been confirmed by biopsy. A similar area was present on the other cheek in which no malignancy developed.

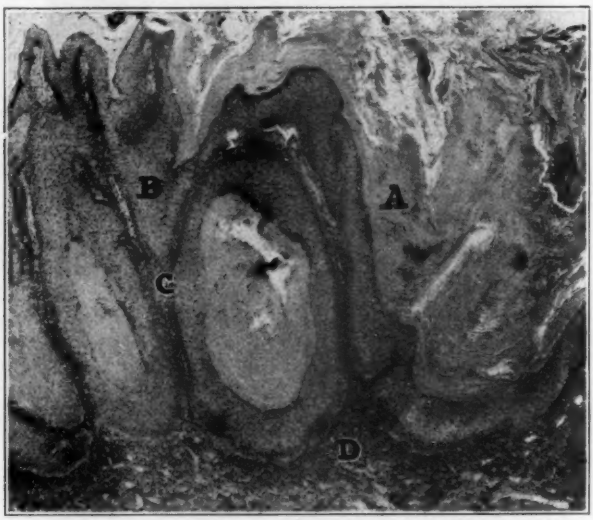

Fig. 5.—Verruca vulgaris. (×18.)

VERRUCA

Under this heading are included those superficial lesions exhibiting acanthosis of the prickle cell layer with accompanying hyperkeratosis. Generally speaking, verruca refers to warty growths not infrequently seen about the lips and cheeks. There are several types of verrucous warts, verruca juvenilis, or "plane wart," and verruca plantaris, seen on the sole of the foot. The verruca vulgaris, or common wart, is the one seen usually in the oral region. However, verruca senilis, or seborrheic keratosis, a lesion more common in older people, is also occasionally seen about the face and lips.

The former (Fig. 5) is characterized by a marked acanthosis with an upward "pushing" of the epidermis. The papillae, consequently, are distorted and appear to be branched or papillomatous. There may be varying degrees of hyperkeratosis. The entire lesion is peculiar in its position above the surface of the skin, the basal layer usually being in its normal relative position. In lesions of long standing, there is an accompanying chronic inflammatory exudate which tends to be perivascular in distribution. Blood vessels may be numerous, with a tendency toward dilation.

Verruca senilis is interesting because of its similarity to basal cell carcinoma. Darier[10] believed it to be nevoid in origin. Some authors have emphasized its non-infectiveness as compared to other types of verruca. Histologically, these warts may have a papillomatous surface with an accompanying acanthosis. (Fig. 6.) The stratified squamous epithelium may show metaplasia toward the basal type with a piling up of these cell clusters on the surface. The amount of hyperkeratosis will vary, but is usually marked and may extend down into the surface crypts. Keratin cysts are a diagnostic feature, as is melanin pigment, which will vary greatly in different lesions. The basal cell clusters with accompanying areas of degeneration account for the similarity to basal-cell carcinoma. Scattered mitotic figures may be seen. This lesion is considered to be more serious than verruca vulgaris, but usually benign.

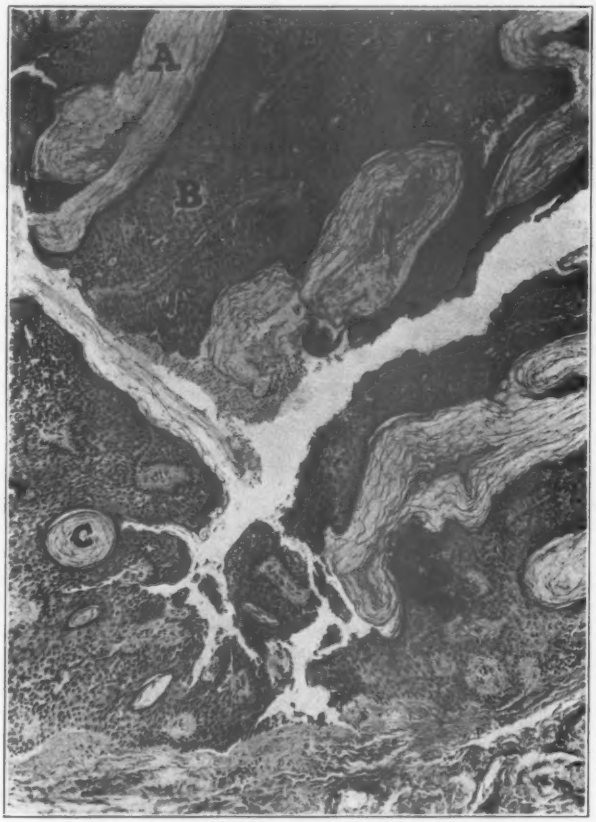

Fig. 6.—Verruca senilis. (×100.)

GRANULOMA PYOGENICUM

Reference is made to this tumor, not because of its importance, either clinically or histologically, but because of its persistent recurrence and lack of response to usual methods of treatment. The earliest references to this lesion con-

sidered it a botryomycetic infection. However, it has been shown that this infection does not occur in man and that the lesion is more likely a granulation response to bacterial infections or trauma. In 1904, Hartzell[11] suggested the name granuloma pyogenicum in preference to numerous other designations, and this name has persisted to date. Wile,[12] in a review of several cases reported, described the lesion as "nothing more than circumscribed granulation tissue," and stated that "its cause was probably *Staphylococcus aureus*, perhaps in an unfavorable soil or in an attenuated form."

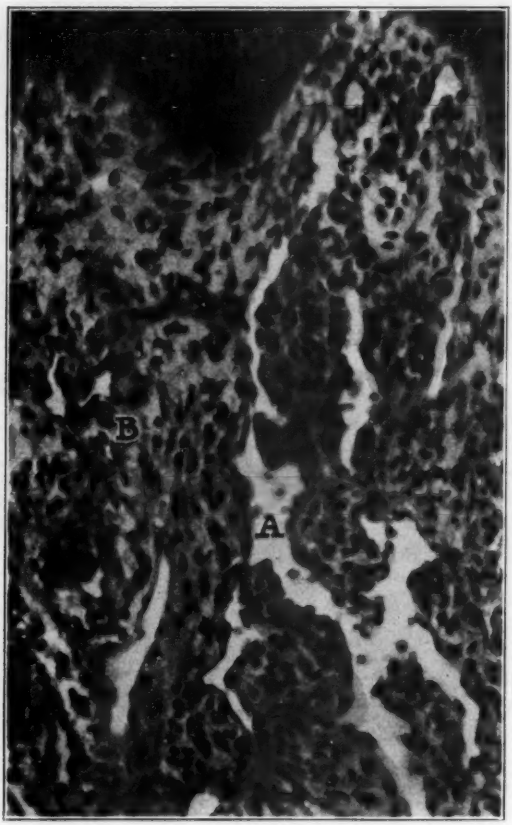

Fig. 7.—Granuloma pyogenicum. (×315.)

It is significant that the lesion occurs about equally in the two sexes and that most cases reported have followed traumatism.

A peculiar red or mulberry color, indicative of its vascularity, characterizes the tumor. Because of its rich blood supply, it bleeds profusely even if but slightly traumatized. Its persistent recurrence indicates that great care must be exercised in its removal.

There is universal agreement on the histology. It is composed of granulation tissue particularly rich in blood supply and new connective tissue fibers. Many

of the vessels may appear as "new" capillaries and others may assume a cavernous appearance, being filled or partially filled with blood. Telangiectasia may be a prominent feature. Obviously, then, certain of these tumors will resemble angioma and the differential diagnosis between the two and gingivitis gravidarum may be quite difficult. The clinical features, however, are usually sufficient to establish its identity. The amount of exudate will also vary, being particularly heavy in those showing areas of ulceration. Certain of the lesions will exhibit a mixed cellular content. Plasma cells are numerous, but not remarkable. (Fig. 7.)

CONCLUSION

In this discussion, those lesions have been considered which have been seen frequently in the routine examination of surgical and biopsy specimens, with resultant interesting problems of differential diagnosis. It must be agreed that a thorough understanding of the pathologic manifestations of these lesions by the operator is one of the most important considerations in their treatment. It is only when the surgeon appreciates this fact that the proper relationship between him and the pathologist can be established.

The diagnosis of any lesion is not a presentation of fact, but rather of opinion, the importance of this opinion being determined by experience and observation. Considering this fact, I have attempted to show some of the numerous variable entities that must be considered in the histologic examination of tissue.

BIBLIOGRAPHY

1. NOVAK, EMIL: Gynecological and Obstetrical Pathology. Philadelphia: W. B. Saunders Co., 1940.

2. TAUSSIG, F. J.: Diseases of Vulva. New York: D. Appleton & Company, 1923.

3. MACLEOD, J. M. H., and MUENDE, I.: Practical Handbook of Pathology of Skin. Ed. 2. New York: Paul B. Hoeber, Inc., 1940.

4. BELL, E. T.: Textbook of Pathology. Ed. 2. Philadelphia: Lea & Febiger, 1934.

5. HAYTHORN, S. R.: Studies on Histogenesis of So-Called "Basal Cell Carcinoma." *Am. J. Cancer*, 15:1969, July 1931.

6. BOYD, WILLIAM: Textbook of Pathology. Ed. 3. Philadelphia: Lea & Febiger, 1938.

7. MACCALLUM, W. G.: Textbook of Pathology. Ed. 4. Philadelphia: W. B. Saunders Co., 1928.

8. FOX, HOWARD: Lichen Planus Confined to Mouth. *Arch. Dermat. & Syph.*, 24:1071, December 1931.

9. SAAD, B.: Squamous Cell Epithelioma Developing on Ulcerated Buccal Lichen Planus. *Bull. Soc. franc. de dermat. et syph.*, 38: 705, April 1931.

10. DARIER, J.: Prec'is de Dermatologie. Ed. 4. Paris: Masson & Co., 1928.

11. HARTZELL, M. B.: *J. Cut. Dis.*, 22:520, 1904.

12. WILE, U. J.: *J. Cut. Dis.*, 27:670, 1910.

EFFECT OF VARIOUS DIETARY DEFICIENCIES ON THE PERIODONTAL TISSUES OF THE GUINEA-PIG AND OF MAN

By Paul E. Boyle, D.M.D.,* Boston, Mass.

DEFICIENCY of several rather than a single dietary essential is the rule when nutritional disease occurs in human beings. Recognition of the rôle played by any one factor is therefore difficult. Fortunately, specific dietary deficiencies may be produced in laboratory animals. In such experiments, a single factor can be eliminated from the diet, and pathologic as well as clinical studies of various stages of the resultant disease be made. Furthermore, the sequences of repair when the specific substance is again added can be studied. By these methods, information of great value has been obtained and, in general, this information has proved to be directly applicable to human beings. Since the periodontal tissues, as well as the skin, hair and eyes, are common sites for the manifestation of deficiency diseases, such experimental studies are of especial interest to periodontists.

The guinea-pig is well suited to nutritional studies. Unlike most other laboratory animals, it is subject to scurvy, the first of the deficiency diseases shown to affect the periodontal tissues. Many other nutritional diseases can also be produced in the guinea-pig. Large numbers of these animals can be kept under rigid laboratory conditions for weeks or months at much less expense than large animals such as dogs or monkeys.

The periodontal apparatus of the guinea-pig is in many respects similar to that of human beings.[1] The teeth are suspended by collagen fibers from the surrounding alveolar bone. Slight movement within physiologic limits is regulated by these suspending fibers as well as by the surrounding fluids and soft tissues. The gingival epithelium is attached to the enamel or cementum. Enamel covers the entire labial surface, including the root of the incisor teeth of the guinea-pig. This makes it possible to separate the suspending part of the parodontium from the non-suspending, purely cushioning region that overlies the enamel. The rate of eruption of the teeth of guinea-pigs is many times that of adult human teeth, but this also is an advantage in that pathologic changes occur much more rapidly. (Fig. 1.)

A complete deficiency of vitamin C (ascorbic acid) in an otherwise normal diet causes the death of guinea-pigs in about four weeks. Loosening of the teeth so that they may be extracted easily is one of the cardinal signs of the deficiency. This loosening results from breaking of the collagen fibers of the periodontal membrane. They may rupture at their attachment to the alveolar bone or the cementum or part way between their attachments. Such failure of the tooth-suspending apparatus is accompanied in the living animal by hemorrhage into

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the periodontal tissues. These are a manifestation of the fundamental consequence of vitamin C deficiency, an inability of connective tissue to form and maintain their normal matrices.[2] (Figs. 2 and 3.)

If vitamin C is administered to a scorbutic guinea-pig, repair takes place with great rapidity. In forty-eight hours, numerous mitotic figures are found in the periodontal membrane, indicating multiplication of the cells that will subsequently produce new suspending fibers. (Fig. 4.) Within a week, the teeth become firmly attached to the alveolar bone.

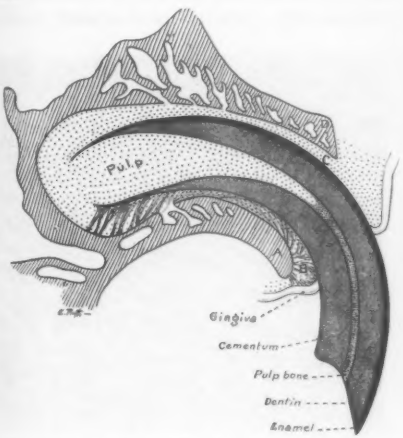

Fig. 1.—Diagrammatic drawing of upper incisor tooth and periodontal apparatus of normal guinea-pig. The dense lingual alveolar bone at A serves as an anchorage for the collagenous fibers (B) of the periodontal membrane. These fibers are embedded in the cementum which covers the concave surface of the incisor tooth. This is the suspending part of the periodontal apparatus. The convex surface of the tooth is covered by enamel and the adjacent periodontal membrane (C) acts as a cushioning apparatus. The labial alveolar bone (D) is relatively delicate.

If amounts of ascorbic acid as low as 2 mg. a day are added to the basal diet, guinea-pigs will grow normally. Histologically, the bones and teeth also remain normal. If the amount of ascorbic acid is reduced to 0.5 or 0.3 mg. a day, many of the animals live for several months, in contrast to the animals receiving no supplement. The incisor teeth of these animals on a minimal dose of ascorbic acid become elongated and displaced labially, while the molar teeth move buccally. Microscopically, it can be shown that the alveolar bone undergoes marked atrophy and that a partial loss of the periodontal fibers, with widening of the periodontal membrane, occurs. (Fig. 5.) These findings indicate that the tooth-suspending tissues are unable to withstand the functional stresses of mastication. They constitute the characteristic syndrome of the systemic or diffuse alveolar atrophy type of periodontal disease.[3]

Fig. 2.—Photomicrograph of suspending apparatus of molar tooth near crest of alveolar bone in normal guinea-pig; detail of collagen fibers imbedded in alveolar bone (A) and extending across periodontal membrane (B) to become attached to cementum of tooth surface (C). The fibers in the periodontal membrane of the incisor tooth follow a more complicated course, but the principle of the suspension apparatus is the same. (Phosphotungstic acid and hematoxylin stain; ×200.)

In adult man, acute signs of scurvy appear only after a much longer deple-

tion period than in the guinea-pig. In a recent experiment,[4] a young physician lived on a diet deficient in vitamin C, but fortified with other vitamins, for a six months' period. At the end of this time, only a minimal tendency toward gingival hemorrhage was observed and little or no increased mobility of the teeth could be demonstrated, although there were definite signs of interruption of the lamina dura.

In scorbutic infants or children, gingival swelling and hemorrhage about the erupted teeth are a universal finding. Synthetic vitamin C causes a dramatic improvement; which is convincing evidence that vitamin C deficiency is the chief etiologic agent. In six instances, postmortem examination of the periodontal tissues revealed changes in the alveolar bone and the periodontal membrane identical with those found in the teeth of scorbutic guinea-pigs. (Fig. 6.)

Fig. 3.—Tissue of scorbutic guinea-pig; location and staining similar to those of Figure 2. The collagen fibers have been ruptured in several places, causing the tooth to become loose. A, alveolar bone. B, broken collagen fibers. C, cementum. (Phosphotungstic acid and hematoxylin stain; ×200.)

Almost nothing is known concerning the effect of long-continued inadequate vitamin C intake on human periodontal tissues. Obviously, diagnosis of inadequacy depends on standards of adequacy. In a recent review[5] concerning the human requirements of vitamin C, Sybil Smith states:

> Values of . . . from 50 to 60 mg. (per day) for adults may be considered as tentative estimates for the middle or barely adequate consumption level, with but little margin of safety or allowance for individual variations in requirement.

This minimum standard is approximately four times as high as that adopted by Radusch[6] in a statistical study of the relationship between periodontal conditions and vitamin C intake (among other dietary factors). Almost one-half the patients in the group most carefully studied by Radusch consumed less than 30 mg. of ascorbic acid daily, a level which she considers "satisfactory plus," but which, according to Smith, is only one-half the "barely adequate" consumption level and

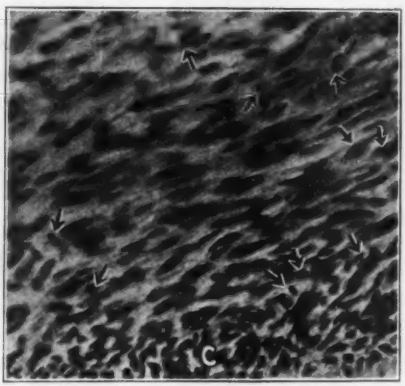

Fig. 4.—Tissue of scorbutic guinea-pig forty-eight hours after administration of vitamin C. Arrows point to mitotic figures in cells which form the collagen fibers of the periodontal membrane. Mitotic figures are less numerous in the periodontal membrane of normal animals and almost entirely lacking in scorbutic animals. C, cementoblasts.

probably about one-third of the optimal level.

The failure of Radusch to find a correlation between periodontal disease and intake of vitamin C may possibly be due to her selection of unduly low standards of adequacy of intake. Furthermore, it is recognized that the therapeutic dose of vitamin C for patients on long-continued inadequate diets may be many times the maintenance dose.[7]

Finally, to judge by the length of time required for manifestations of signs of the complete deficiency of vitamin C in human beings, partial deficiency would not become clinically evident in the periodontal tissues for months or years.

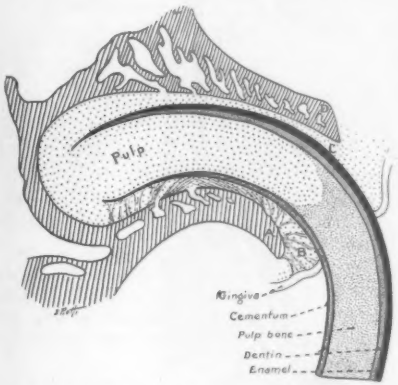

Fig. 5.—Diagram of upper incisor tooth and periodontal apparatus of guinea-pig maintained for 100 or more days on inadequate amounts of vitamin C. The tooth has been completely renewed during the experimental period. A narrow layer of essentially normal dentin continues to be formed at the proximal part of the tooth and persists throughout the length of the incisor, but the bulk of the tooth is composed of irregularly formed pulp bone. The alveolar bone (A) is irregularly shaped, the periodontal membrane is wide and the suspending collagen fibers (B) are less numerous than normal. C, labial periodontal membrane. D, labial alveolar bone.

Trauma and infection which cause lesions in a relatively short period tend to obscure evidence of the effects of vitamin deficiency. Dietary histories stating that the intake of vitamin C was satisfactory in many "pyorrhea" patients can have little bearing on this point unless "pyorrhea" due to local conditions is ruled out and only systemic or diffuse atrophy "pyorrhea" is considered.

In mouth conditions of patients in whom careful dietary histories indicate a long-continued deficiency of vitamin C confirmed by low blood-plasma, ascorbic acid levels are more valid evidence. Here also, the effects of local trauma are difficult to exclude, but preliminary observations, including therapeutic response to ascorbic acid therapy, indicate that vitamin C deficiency is an important cause of periodontal disease of the systemic type.[8] Furthermore, pathologic examination of the periodontal tissues of patients known to have had suboptimal intakes of vitamin C for long periods reveals atrophy of the alveolar bone, widening of the periodontal membrane and partial loss of fiber attachment similar to that found in guinea-pigs.

Vitamin A deficiency has been shown to affect certain epithelial tissues and to be necessary for the formation of visual purple and for normal growth of bones and teeth.[9] Guinea-pigs, unlike dogs,[10] show little or no increased tendency to periodontal infection with pocket formation when kept on diets deficient in vitamin A. Atrophy of the alveolar bone and retardation of new bone formation are found. Although vitamin A deficiency is known to occur in human beings, no report of its effect on the periodontal tissues is available.

Diets low in calcium and vitamin D, but adequate, as far as is known, in all other respects, cause typical rickets in young, rapidly growing guinea-pigs.[11] The alveolar bone shows characteristic osteoid margins on the bone trabeculae. The lingual alveolar bone, to which the bulk of the suspending fibers of the periodontium are attached, often becomes

bent similarly to the bowing of the legs in human rickets. (Fig. 7.)

No pathologic studies have been made of the alterations in human periodontal tissues which may occur as the result of diets low in calcium and vitamin D. There is much evidence, however, that this is one of the common deficiencies in the diets of civilized man.

As one type of control for the experiments described above, guinea-pigs have been kept on diets that are adequate in all known respects, including a liberal intake of vitamins, but the amounts of which were greatly restricted. Such animals nearly starve. The periodontal tissues, however, remain essentially normal.

Fig. 6.—Maxillary deciduous central (I) and permanent central (1) and lateral (2) incisor teeth of 11-months-old scorbutic infant. The lingual (A) and labial (B) alveolar bone show marked rarefaction with widening of the periodontal membrane. (Hematoxylin and eosin stain; ×11.)

SUMMARY

1. Controlled nutritional studies utilizing the guinea-pig as an experimental animal indicate that:

(a) The periodontal tissues are not affected specifically by starvation.

(b) Vitamin A deficiency causes retardation of bone formation, including the alveolar bone, but does not predispose to gingival infection with pocket formation.

(c) Vitamin D deficiency, with a low

intake of calcium, causes typical rickets in young animals, with characteristically defective calcification of the bones, including the alveolar bone, and the latter becomes bowed under the pull of tooth suspension.

(d) Complete lack of vitamin C causes rupture of the periodontal fibers with hemorrhage; addition of vitamin C brings about rapid repair with reformation of the periodontal fibers.

(e) An inadequate intake of vitamin

Fig. 7.—Longitudinal section of upper incisor tooth of rachitic guinea-pig. The lingual alveolar bone (A) is bent in the direction of the pull of the suspending fibers. (Hematoxylin and eosin stain; ×4.5.)

C over relatively long periods results in inability of the periodontal tissues to withstand functional stress with migration of the teeth, which are the characteristic features of the systemic or diffuse atrophy type of periodontal disease.

2. Limited clinical and pathologic studies indicate that vitamin C deficiency is a frequent cause of the systemic type of periodontal disease in human beings.

BIBLIOGRAPHY

1. BOYLE, P. E.: Tooth Suspension. Comparative Study of Paradental Tissues of Man and of Guinea-Pig. *J. D. Res.*, 17:37, February 1938.

2. WOLBACH, S. B., and HOWE, P. R.: Intercellular Substances in Experimental Scorbutus. *Arch. Path. & Lab. Med.*, 1:1, January 1926.

3. BOYLE, P. E.; BESSEY, O. A., and WOLBACH, S. B.: Experimental Production of Diffuse Alveolar Bone Atrophy Type of Periodontal Disease by Diets Deficient in Ascorbic Acid (Vitamin C). J.A.D.A., 24:1768, November 1937.

4. CRANDON, J. H.; LUND, C. C., and DILL, D. B.: *New England J. Med.*, 223:353, September 5, 1940.

5. SMITH, S. L.: Human Requirements of Vitamin C. *J.A.M.A.*, 111:1753, November 5, 1938.

6. RADUSCH, D. T.: Relationship Between Periodontal Conditions and Certain Dietary Factors. *J. D. Res.*, 18:305, August 1939.

7. ABT, A. F., and FARMER, C. J.: Vitamin C. Pharmacology and Therapeutics. *J.A.M.A.*, 111:1555, October 22, 1938.

8. WEISBERGER, DONALD; YOUNG, A. P., and MORSE, F. M., JR.: Ascorbic Acid Blood Levels in Dental Patients. *J. D. Res.*, 17:101, April 1938.

9. BESSEY, O. A., and WOLBACH, S. B.: Vitamin A. Physiology and Pathology. *J.A.M.A.*, 110:2072, June 18, 1938.

10. MELLANBY, MAY: Diet and Teeth. Part II A. London: Medical Research Council, 1930.

11. HOWE, P. R., et al.: Low Calcium Rickets in the Guinea-Pig. *Proc. Soc. Exper. Biol. & Med.*, 45:298, October 1940.

25 Shattuck Street.

THE MAXILLARY ANTRUM AND ITS DENTAL ASPECTS

By Ray Parker, M.D., Johnstown, Pa.

I APPRECIATE any opportunity which permits me to urge greater cooperation between our professions. Under the pressure of immediate concerns, and we are busy people, we have not had sufficient opportunity to interchange professional interests and knowledge. This grieves me, for the medical man would be a better physician if he had more knowledge of the teeth, and some knowledge of general medicine would surely be an aid to the dentist.

Several years ago, we had a combined meeting of our professional societies. The speaker was Robert H. Ivy, professor of maxillofacial surgery at the University of Pennsylvania's postgraduate school. The subject was of common interest and the whole meeting tended to bring us together. We need a more coordinated attack in our work, since our professions overlap, and especially is this true of the dentist and the eye, ear, nose and throat specialist.

The maxillary antrum, especially, is common ground for medicine and dentistry. Sooner or later, every dentist meets antral disease. Sometimes it involves the teeth secondarily, but, more often, when both teeth and sinus are involved, the process begins in the teeth. Some writers say that the teeth start the infection only 2 per cent of the time. At the other extreme, there are a few who say that antral disease begins in the teeth always. The truth probably lies between, the commonly accepted percentage being 25 or 30.

Read before the Cambria County (Pennsylvania) Dental Society, February 24, 1941.

The maxillary antrum, as you know, is situated in the upper jaw bone, and follows in the main the shape of the body of this bone. At birth, it is simply a slitlike cavity 8 by 4 by 6 mm. In the adult, it is irregularly pyramidal and measures about 34 by 32 by 23 mm. There are, of course, many variations. The shape is influenced by the shape of the nasal chambers, the thickness of the sinus walls, the encroachment of the facial wall, etc. A prominent malar eminence with a deep canine fossa makes a difficult surgical approach. Occasionally, the antrum is divided into subcompartments. Sometimes, a posterior ethmoid cell encroaches on the maxillary sinus.

The opening from the antrum into the nose, the ostium as it is called, is of a good deal of importance since on the size and position of this opening depend the passage of air into the antrum when we breathe and the passage from the antrum of any discharges which might accumulate as a result of disease. In the first place, the ostium is disadvantageously placed as a drainage aperture since it is located at the extreme upper part of the sinus and opens into the nose under the middle turbinate bone. Any nasal congestion swells the mucous membrane lining in the nose, and when that covering the middle turbinate swells, it may completely close the opening into the antrum, with consequent loss of both ventilation of and drainage from this sinus. The ostium always is in the forward part. It varies in size and shape from a tiny round aperture to a long

slotlike opening. The most usual form is a round opening about the size of a buckshot. In about 10 per cent of cases, there are accessory ostia.

The number of teeth bearing direct relation to the antral floor varies. Those most constantly found are the three molars and the second premolar. When the sinus is small, the second and third molars only may be in direct relation to the antral floor.

It was formerly thought that all antral disease was due to carious roots. The abandonment of this idea is no doubt due to better diagnosis on the part of the nose and throat man. Irrigation of the antrum is a common office procedure, or at least it should be. It is the best diagnostic procedure we have. There is hardly a day that this is not done in my office at least once. The laity have strange ideas of what they usually term draining the antrum. The usual procedure is to cocainize the lateral wall under the inferior turbinate and then pass a small straight hollow needle through this wall. I prefer a Litchwich needle for this. The antral wall is frequently very thin at this point, and little discomfort is caused. The dangers are slight. After the needle is inserted, the stylet should be passed through to make sure that the open end is free in the antrum. Then one should carefully and slowly inject a syringe full of air, and if there is obstruction, stop. This means that the end of the needle is in the mucous membrane lining or in a polyp or tumor, etc., and the treatment is halted for investigation in other ways. If the air goes in easily, I then irrigate with a warm physiologic solution of sodium chloride. The irrigating fluid goes in through the needle at the bottom of the antrum and washes out the ostium at the top. If the washings contain pus, there is no doubt that a purulent sinusitis is present. It is my practice in these cases to irrigate daily until definite improvement is obtained, but irrigation is not entirely discontinued until the irrigating solution returns clear.

Formerly, large caliber curved trocars were used, and this was usually a very painful procedure. Needless to say, the needle should always be sharp and smoothly nickeled. When rusted or dull, they should be discarded.

In my own experience, the possibility of dental disease causing trouble in the antrum varies a great deal in individual subjects. The distance between the apical portion of the roots and the floor of the sinus is inconstant, depending upon the amount of cancellated bone structure which intervenes. Certain specimens show the distance separating these structures to be several millimeters, while others show the roots projecting into the sinus.

In some instances, the sinus mucosa alone separates the root from the cavity proper. The interior of the maxillary sinus is not always smooth, but quite often presents partial septa. These are usually situated on the floor and in the superior internal angle. There are some abnormalities of the antrum as to size and shape, the principal reason for this being resorption of bone during the formation of the sinus.

The antral lining is important. It is a mucous membrane, the outer layer of which consists of ciliated epithelium. Next to the bone is the periostium with connective tissue between. The lining of the antrum rarely exceeds 0.02 mm. in thickness. It is somewhat loosely attached about the ostium and prone to edematous swelling on slight irritation. The ciliated epithelium, of course, tends to expel accumulating discharges. In this way, if the infection is not too severe, the antrum can take care of its own drainage. The blood supply of the bony antral wall is double, both sides being covered with periostium, through which the bone receives nutrition. This explains

why severe infection of the mucosa is usually unaccompanied by disease of the underlying bone.

The antrum is more often diseased than any of the other sinuses, supposedly owing to its poor drainage and to the proximity of the dental roots. The fact that the antrum is the sinus most frequently diseased is fortunate for the patient because no other sinus is so accessible.

There are many causes of antral infection, such as infection from the nose proper or from some other sinus, disease of the bone, such as osteomyelitis, tuberculosis, syphilis and malignancy, but the question which probably bothers the dentist is simply whether the patient has antral disease. If his trouble is entirely dental, it is one thing, but if it is a combination of dental and antral disease, the problem is entirely different. Of course, no one can make correct diagnoses 100 per cent of the time, and I have no magic formula by which one can always tell the condition of the antrum. There are several things that are an aid in judging individual cases. If I were to have only one thing from which to make my diagnosis, I would take the history. Remember this—sinus trouble is nose trouble, and if the patient has no nasal symptoms whatever, he has no disease in any of the sinuses.

The patient with sinus disease must have some abnormality of the nasal function. There will be an increased nasal discharge and, in getting the history, we must remember that a nasal discharge may be forward from the nostrils or back into the pharynx (postnasal discharge.) Some people who have chronic infections have developed the symptoms slowly over a considerable period of time and do not realize how much discharge is present. I usually ask how many handkerchiefs are used daily. If the trouble in the nose dates to a head cold, which apparently had never gotten better, we have another point in favor of sinus infection. If the patient states that there is an offensive odor to that nasal discharge, retention of secretions is suggested.

The symptom of pain, I think, is an unreliable one. I kept records for a while on this question of pain in relation to antral disease. In all the cases, irrigation yielded quantities of visible pus. An enameled basin colored black inside is most satisfactory for this purpose as foreign material shows up much more plainly. I do not remember the exact percentages and have been unable to find my records, but they were approximately as follows: 20 per cent had no pain; 50 per cent had frontal pain; 10 per cent had occipital pain, and only 10 per cent had pain over the antrum, and in 10 per cent all the pain seemed to be in the teeth. In many of the chronic cases, there was no pain.

The nose is surely obstructed if there is too much discharge. However, there may not be much discharge and still obstruction be present. This occurs in the cases which are mild and in which, while there is little increase in discharge, there is congestion enough in the nasal mucosa to cause obstruction. Transillumination is always mentioned as a diagnostic aid, but I have never found it a very reliable one. To employ transillumination, one should have a dark room. The findings are always influenced by the thickness of the cheek, bone, etc., and one can only make a deduction by comparing both sides. What applies to transillumination also applies to the x-rays. There are so many structures throwing shadows on the film that it is very difficult to separate them and make a diagnosis from this source only.

The cases of antral disease following dental infection are better understood by you than by me. Removal of a tooth the roots of which extend into the antrum I do not think can of itself cause antral disease. Where antral infection is present, following extraction, a consider-

able area of the antral lining is also diseased. I do not think this can be cured by irrigation through the socket of a tooth root, at least not usually. I have one patient in mind, a woman aged 30 who was referred to me by one of your members. She had had an extraction with an opening into the antrum seven months previously and persisting antral symptoms: headache and foul discharge both from the nose and into the mouth. As irrigation had no effect, after a fair trial I abandoned this line of treatment and performed a radical antrum operation. The Caldwell-Luc operation is best suited to this type of a case. This was a case of antral disease secondary to dental disease. Another patient, aged 27, complained of pain in the left upper jaw, which she felt was coming from a tooth. Her dentist had referred her to me, saying there was no dental disease. She was angry at the dentist because of this decision, insisting that she had toothache. Irrigation yielded a large amount of pus, however. A Caldwell-Luc operation was necessary to clear up the condition. The teeth were not involved, this being a case of primary disease of the antrum with dental symptoms.

One meets odd cases. My star case was that of a woman aged 27 with all the symptoms in the book. She had made the rounds and was put down generally as a neurasthenic. Since she complained of many nasal symptoms, I irrigated the antrum. I thought that I had made a spectacular cure when the antrum was clear after several days of irrigation; but in about two weeks the patient was back with all the symptoms again. Irrigation again cleared up the antrum, but the patient came back a third time. She had no upper teeth on the side affected, but I was a little suspicious by this time and directed her to her dentist, who said that the teeth she had were healthy. To be on the safe side, he roentgenographed the area under the antrum where there were no teeth. The films were all negative but one, which showed a peculiar shadow of the floor of the antrum. Since little of the antral floor was shown on the dental film, the dentist suggested that I have some x-ray studies of the antrum made. This was done, the report coming back "normal antrum." The symptoms persisted, however, so I finally performed an external operation. When the antrum was opened, the odor was very offensive and on the antral floor was a good-sized root. The patient told me afterwards that the teeth in that area had been extracted eighteen years before. She still has many symptoms, but not of antral disease.

A root in the antrum is, of course, a foreign body and should be removed. Such a foreign body has been removed by irrigation, but it seems a long chance. The Caldwell-Luc operation has always given great satisfaction. I have performed it under local anesthesia for several years. The patient is given a regular breakfast, entering the hospital around 10 a.m. At noon, a soft diet is given and, shortly after, a barbiturate. At 1 p.m., a hypodermic of morphine and atropine is given, with the operation scheduled for 2 p.m. I usually cocainize the region of the inferior turbinate and then inject 5 cc. of procaine (0.5 per cent) into the sphenopalatine (Meckel's) ganglion. I used to do this, or at least I tried to, as you men do by entering back of the last tooth.

For the last two years, I have used the external approach, introducing the needle just under the zygoma, using a 42 mm. needle and withdrawing the plunger a little at first to make sure that a blood vessel has not been entered. I am not so accustomed to working in back of the teeth as the dentists, and one can be a little more sure of sterilization of the skin. I drop a line from the posterior orbital ridge to the lower border of the zygoma. First, a drop or two of the solution is injected into the skin, then the needle is advanced, upward

and backward slightly, keeping against the posterior surface of the maxilla.

One may have to manipulate the needle a little, and after the 42 mm. stop is against the skin, the solution is injected slowly. I always insert the needle a little at a time, withdrawing the plunger each time before advancing farther. The needle now is close to the ganglion. By blocking the maxillary nerve (second branch of fifth), there is anesthesia of the cheek, part of the face, nose, lip, upper teeth, lateral wall of the nose, antrum, soft palate, tonsils and roof of the mouth. The only other injection made is infiltration over the site of the incision. I infiltrate over the canine fossa where I make the incision and, after retracting the periosteum, I use a drill or a trephine to make the opening into the bone.

If the operation is to remove a foreign body only, I do not disturb the antrum further. If the antrum is diseased, I remove the diseased mucosa by wiping it off with gauze moistened with a solution of picric acid in acetone. This removes all diseased mucosa and does not encourage bleeding. It also removes the danger of any possible damage with the curet. In these cases, I also make a counter opening into the nose. I use general anesthesia only if the patient is extremely nervous. Under local anesthesia, there is little bleeding; which means shorter operating time, and, therefore, less reaction, usually permitting the patient to leave the hospital within forty-eight hours. I do not put sutures in the incision over the canine fossa, as the cheek keeps the edges of the wound together and healing takes place rapidly.

As to what a dentist should do if a root escapes into the antrum, that is a question which he has to decide for himself. I do feel that the root should not be left there. With your familiarity with the mouth, you need have no fear in opening the canine fossa. The chances of getting the root out by way of the tooth socket seem very slim to me. If you do not remove it, however, it is best to tell the patient about it, with instructions to come back if symptoms arise suggesting infection, with odor and nasal discharge.

I have been asked what I would do if, during an extraction I felt the antral floor, or a large part of it, giving way. If it were a good-sized area, I would try to dissect the tooth out, being as careful as possible not to separate the bony antral floor from its periosteum and not to lacerate the mucous membrane of the mouth. If the mucous membrane and the periosteum remain attached to the bone, the chances are that the bone will reestablish union just as if there had been a fracture. Immobilization might be obtained by the use of a partial denture or some device attached to the teeth. You have the ingenuity to solve that part of the problem. Considering the possibility that a part of the antral floor has been torn away, mucous membrane and all, one would have somewhat the same state of affairs as in cleft palate. There would surely be disturbance of speech, and, of course, there would be regurgitation of food into the nose. This problem might be solved by operative measures similar to those used in closure of a cleft palate, or, again, a denture might answer. Each case would, of course, present features requiring individual consideration.

907 United States Bank Building.

STUDIES ON THE RELATIONSHIP BETWEEN DIASTATIC ACTIVITY OF SALIVA AND INCIDENCE OF DENTAL CARIES

By H. J. Florestano, Ph.D.; J. E. Faber, Ph.D., and L. H. James, Ph.D., College Park, Md.

CONSIDERABLE attention has been given to the study of physicochemical properties of saliva in an attempt to find some factor that might explain or at least be correlated with the development of caries in teeth. Hubbell[1] found the blood-serum calcium, the inorganic acid-soluble phosphorus, the carbon-dioxide capacity and pH of the blood to be the same in both carious and non-carious individuals. A difference in the carbon-dioxide capacity and titratable alkalinity of the saliva, however, was correlated with dental caries. Marshall[2] likewise observed a relationship between the titratable alkalinity of the saliva and caries susceptibility. Most observations on the pH of the blood and of the saliva of carious and non-carious individuals have shown such little differences that they become practically valueless as criteria of individual susceptibility. Karshan, Krasnow and Krejci[3] found a relationship between the pH and caries, but believed the titratable alkalinity to be more reliable.

It is apparent from a review of the literature that ptyalin has received little attention as a possible factor in dental caries. Gore[4] has shown that salivary ptyalin is capable of hydrolyzing the complex carbohydrate moiety of mucin into simpler sugar, which is thus made available for fermentation to lactic acid by acidogenic bacteria present in nearly all mouths. He found that the amount of carbohydrate combined with the protein in mucin varied greatly, and that the time required for its hydrolysis to fermentable sugar was in direct proportion to the concentration of ptyalin in the saliva. Examining samples of saliva from carious and non-carious individuals, Gore observed that the carbohydrate content was always higher in the former, and that when the carbohydrate intake was increased, diastatic activity increased. Hubbell[1] analyzed saliva of fifteen non-carious and seventeen carious children and found no consistent differences in diastatic activity between the two groups.

In the present investigation, a number of individuals were studied in order to compare the ptyalin content of saliva with the incidence of caries.

EXPERIMENTAL DATA

Subjects.—The majority of subjects used in the investigation consisted of inmates of the District of Columbia Penal Farm, Lorton, Va. The remainder were patients of private practitioners. A total of 166 persons were studied. Each subject was given a thorough dental examination and his case history was recorded. Of the total number of cases, seventy-six were non-carious and ninety were carious. The individuals were divided into the following eight groups according to their dental conditions:

I. Non-carious:

Group A. Those with all teeth intact and exhibiting no open cavities or fillings (twenty-eight individuals).

Group B. Those with previous extrac-

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tions, but no existing cavities or fillings (twenty-nine individuals).

Group C. Those with no open cavities, but with teeth filled (nineteen individuals).

II. Carious:

Group D. Those with one open cavity; designated as first degree caries (nineteen individuals).

Group E. Those with two open cavities; second degree caries (ten individuals).

Group F. Those with three open cavities; third degree caries (five individuals).

Group G. Those with from four to ten open cavities; fourth degree caries (twenty-eight individuals).

Group H. Those with ten or more open cavities; rampant caries (twenty-eight individuals).

Collection of Saliva.—A determination of the diastatic activity was made on every sample of saliva obtained from each subject. Increase in flow of the saliva was induced by chewing sterile paraffin, and approximately 25 cc. of saliva was collected from each subject in sterile sputum bottles. Saliva specimens were collected between 10 a.m. and 12 m. As soon as samples were obtained, they were packed in an iced container for transportation to the laboratory. An interval of about three hours occurred between the time of collection and the time of analysis. The starch-iodine test was used to determine diastatic activity.

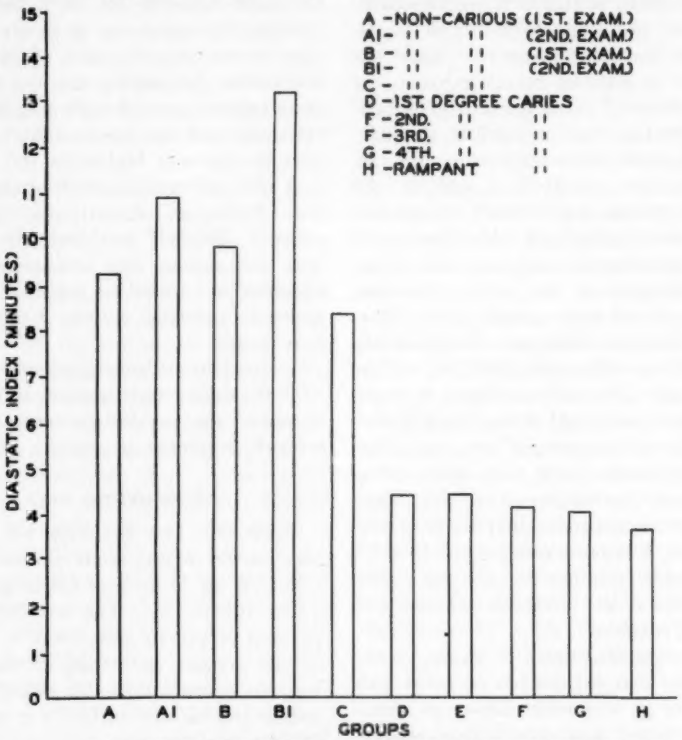

FIG. 1. AVERAGE DIASTATIC ACTIVITY OF SALIVA OF NON-CARIOUS AND CARIOUS INDIVIDUALS

Starch Solution.—A 1 per cent solu-

tion of starch was prepared by dissolving soluble starch powder (Baker's) in distilled water. The solution was boiled for fifteen minutes, cooled to room temperature and then restored to the original volume by the addition of distilled water.

Iodine Solution.—A 2 per cent solution of potassium iodide was prepared in distilled water and the solution colored a deep yellow by the addition of 10 mg. of iodine crystals (resublimed).

The starch and iodine solutions were never more than 2 days old, and when not in use were kept at 4-5° C.

Conditions for the tests were carefully standardized. Solutions were thoroughly agitated before use. Saliva samples were shaken for one minute and passed through filter paper before testing. With a pipet, 20 cc. of starch solution was measured into an 8 by 1 inch test tube and placed in a waterbath at 37° C. When the starch solution reached the temperature of the bath, 5 drops of the filtered saliva was introduced into the solution, and the time required for complete hydrolysis of starch was determined by the spot-plate method. The changing of the mixture of starch and iodine from light brown to yellow was taken as an indication of complete hydrolysis and served as the diastatic index of the saliva.

TABLE 1.—DIASTATIC ACTIVITY OF SALIVA OF NON-CARIOUS INDIVIDUALS

Group A		Group B			Group C		
Case	D.I.*	Case	No. of Teeth Missing	D.I.	Case	No. of Teeth Filled	D.I.
38	10.5	12	4	21.5	1		6.5
42	10.0	41	14	7.5	5		4.5
75	28.5	62	3	24.5	7	4	8.5
96†	2.5	63	4	7.5	8	4	9.5
102†	2.5	65	4	31.0	9	8	7.0
124	4.5	66	8	20.0	11	3	13.5
138	4.5	67	3	14.0	13	3	11.0
139	3.0	71	5	11.5	23	6	8.5
140†	4.5	73	7	7.5	31	3	7.5
148	4.0	79†	2	4.5	33	2	9.5
149	8.5	83†	2	2.0	51	2	6.5
150	10.0	86†	6	2.5	52	4	6.5
151	26.0	88	2	3.5	56	1	14.0
152†	5.5	93	7	6.0	57	6	12.5
153†	4.5	94	24	26.0	60	3	5.0
154†	5.5	95	4	5.0	68	2	2.5
155	6.5	97	7	7.5	72	3	4.5
156	7.5	104	2	8.5	76	4	9.5
157†	5.0	107†	2	4.5	77	11	12.0
158	6.5	111†	6	3.5			
159	12.5	114†	11	2.5			
160	15.5	115†	11	2.5			
161	16.5	117	2	6.0			
162	16.0	120†	3	3.0			
163	7.5	126†	5	4.5			
164	7.5	128†	6	4.0			
165	11.0	130	1	27.5			
178	13.5	136	1	2.5			
		146	8	13.0			
Average D.I.	9.3			9.8			8.4

*Diastatic index; time in minutes for complete starch hydrolysis.
†Individuals developing caries within ten months after initial examination.

RESULTS AND DISCUSSION

The diastatic index for each individual is shown in Tables 1 and 2. In general, the time required for starch hydrolysis was greater in non-carious than in carious cases. The average time for complete diastatic action of the saliva in all seventy-six non-carious cases was 9.3 minutes; while, for the ninety carious cases, it was 4.0 minutes. It may be observed in Table 1 that for the twenty-eight immune individuals (Group A), the average diastatic index was 9.3 minutes; whereas, for the twenty-eight individuals with rampant caries (Group H), the average time for complete starch hydrolysis was 3.6 minutes. An increase in diastatic activity was accompanied by an increase in the degree of dental caries. (Fig. 1.)

It was found that, in the non-carious groups (Table 1), a number of individuals possessed diastatic indices comparable to those in the carious groups (Table 2). This was especially noticeable in Groups A and B. It was not practicable to reexamine all subjects included in the study. However, periodic examinations were subsequently made on members of the two non-carious groups to see whether caries developed in those individuals having a high diastatic activity. Of eleven men in Group A with low diastatic indices ($2\frac{1}{2}$-$5\frac{1}{2}$ minutes), seven, or 64 per cent, developed caries within ten months after the initial ex-

TABLE 2.—DIASTATIC ACTIVITY OF SALIVA OF CARIOUS INDIVIDUALS

Group D		Group E		Group F		Group G		Group H	
Case	D.I.*	Case	D.I.	Case	D.I.	Case	D.I.	Case	D.I.
44	4.0	30	5.0	10	3.5	2	4.0	3	1.5
49	5.0	48	8.0	53	3.5	14	1.5	4	1.5
61	5.0	87	2.5	59	8.5	18	4.5	6	2.5
78	9.5	90	11.0	135	1.5	21	6.0	15	3.5
80	2.5	108	2.5	137	4.0	24	4.5	16	1.5
84	3.5	118	3.5			26	7.5	17	2.5
89	13.0	121	4.5			27	5.0	19	5.0
92	4.5	134	1.5			28	3.0	20	4.0
103	3.5	143	3.5			34	2.0	22	6.5
106	5.5	145	1.5			35	2.5	29	2.5
116	3.0					40	2.5	32	3.5
119	3.0					43	3.5	37	1.5
122	3.5					45	5.0	46	5.5
123	3.0					47	5.0	58	4.5
131	4.5					50	5.0	69	4.5
132	3.5					55	3.5	129	7.0
141	1.5					64	4.5	166	4.0
144	3.5					70	4.5	167	3.0
147	1.5					74	7.0	168	3.5
						98	5.0	169	6.0
						101	3.5	170	5.0
						105	3.0	171	1.5
						112	3.5	172	2.5
						113	2.0	173	3.0
						125	10.0	174	1.5
						127	2.5	175	5.5
						133	2.5	176	3.0
						142	2.0	177	5.5
Average D.I.	4.4		4.4		4.2		4.1		3.6

*Diastatic index; time in minutes for complete starch hydrolysis.

amination. In Group B, thirteen men originally had comparatively high diastatic activities, of which ten, or 77 per cent, developed caries within ten months. When the diastatic indices of those individuals of Groups A and B who developed caries within ten months were not included in the calculations, the average time increased from 9.3 to 10.7 minutes for Group A (A-1, Fig. 1) and from 9.8 to 13.2 minutes for Group B (B-1, Fig. 1). These observations seem to indicate a close relationship between the diastatic activity and the development of dental caries. The negative results obtained by Hubbell[1] may be explained by the fact that too few cases were studied.

Some of the saliva samples used in these tests were obtained from inmates of the prison farm during the course of a study on the influence of grapefruit upon oral hygiene and general health. Thus, some of the subjects providing the samples were examined by the resident dentist periodically, and all information compiled on this group during the study showed a definite improvement in health. Therefore, any influence of the grapefruit diet upon these studies would have been to reduce rather than to increase dental caries.

From the results presented, it is believed that the diastatic activity of saliva may serve as an index of caries susceptibility. Further, there probably is a direct relationship between diastatic activity and the growth of lactobacilli in the mouth.

Oral aciduric bacteria require simple sugars for acid production, and it is possible that, in this respect, diastatic activity may be an essential factor. By virtue of the action of ptyalin on complex carbohydrate, simpler sugars are formed and thus made available for acid fermentation by aciduric organisms, which are almost invariably present irrespective of the incidence of dental caries. This may aid in explaining why Jay[5] has been able to control caries in some cases by restricting sugar alone, while, in other cases, both sugar and starch had to be restricted.

SUMMARY AND CONCLUSIONS

Saliva from 166 subjects was examined for ptyalin content, with the starch-iodine technic. Oral examination at the time the saliva samples were collected permitted the segregation of the subjects into eight groups, namely: (A) non-carious, no teeth filled nor missing, (B) non-carious, no teeth filled, teeth missing, (C) non-carious, teeth filled, (D) first degree caries, (E) second degree caries, (F) third degree caries, (G) fourth degree caries and (H) rampant caries.

The average diastatic index of each group showed a direct relation to the incidence of caries, the greater diastatic activity being accompanied by a higher incidence of tooth decay.

The average diastatic index of individuals with rampant caries was three times that of individuals showing no caries.

It is suggested that the diastatic activity of saliva be used as an index of caries susceptibility.

G. W. Bogikes, of the District of Columbia Penal Farm, Lorton, Va., and W. L. Smallwood and Sterling V. Mead, Washington, D.C., cooperated in the selection and examination of dental patients used in this study.

BIBLIOGRAPHY

1. HUBBELL, REBECCA B.: Chemical Composition of Saliva and Blood Serum of Children in Relation to Dental Caries. *Am. J. Physiol.*, 105:436, August 1933.

2. MARSHALL, J. A.: Composition of Saliva and Dental Caries. *Am. J. Physiol.*, 43:212, 1917.

3. KARSHAN, MAXWELL; KRASNOW, FRANCIS, and KREJCI, LAURA E.: Study of Blood and Saliva in Relation to Immunity and Susceptibility to Dental Caries. *J. D. Res.*, 11: 573, August 1931.

4. GORE, J. T.: Saliva and Enamel Decalcification. *D. Cosmos*, 77:942, October 1935.

5. JAY, PHILIP: Rôle of Sugar in Etiology of Dental Caries. J.A.D.A., 27:393, March 1940.

FACTORS IN THE ETIOLOGY OF MOTTLED ENAMEL

By Floyd DeEds, Ph.D., San Francisco, Calif.

INTEREST in the chronic toxicity of fluorine had its origin in the problems confronting four groups of investigators. Three of these groups were directly concerned with the public health aspects of exposure to fluorine and fluorine compounds, while the fourth group was more immediately interested in the effects of feeding phosphate rock to cattle as a mineral supplement to the diet.

The data secured by all four groups of investigators have contributed to our understanding of chronic fluorine poisoning and, in particular, its relation to the production of mottled enamel. Two reviews on fluorine poisoning published in 1933[1, 2] contained about 120 references each, and the monograph published by Kaj Roholm[3] in 1937 listed 893 references. At the present time, the list of contributions to the subject is even greater. Due credit for the contributions made by various investigators is given in the three above-mentioned publications. Therefore, in the interest of conserving space, references will generally be omitted in the summary of the basic facts regarding fluorine toxicity. This summary will be followed by a discussion of recently demonstrated factors in the production of mottling of enamel, and by evidence that ingestion of cadmium can produce a similar, if not identical, enamel defect.

From the Agricultural Chemical Research Division, Bureau of Agricultural Chemistry and Engineering, United States Department of Agriculture, at the Department of Pharmacology, Stanford University School of Medicine.

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EARLY STUDIES

The studies of the first group of investigators were directly concerned with the relation of mottling of enamel to drinking water and in particular to its fluorine content. The second group of workers was interested in the chronic toxicity of fluorine-containing insecticides used as substitutes for sprays of lead arsenate. A third group of investigators was interested in chronic fluorine poisoning because people and livestock were subjected to the fluorine gas discharged in certain industries, and workers engaged in the mining and grinding of cryolite, sodium aluminum fluoride, were subjected to an industrial hazard. Phosphate rock containing varying amounts of fluorine is used as a mineral supplement in feeding cattle, and consequently the investigators in various agricultural experiment stations constituted a group interested in the general problem of fluorine toxicity.

Perhaps the most extensive data on and most conclusive evidence of the toxicity of fluorine for man concern the presence of mottled enamel in persons indigenous to certain endemic areas, where the severity of the enamel defect is correlated with the fluorine content of the drinking water. Drinking of these waters or the administration of sodium fluoride in concentrations equivalent to that in the water has been shown to cause bleaching of albino rat incisors.

The wide distribution of these areas

where mottling of enamel is endemic in the United States is shown in Figure 1. The world-wide distribution of such areas is shown in Figure 2. Doubtless, the areas are even more numerous than here indicated, but surveys are lacking to prove this. That fluorine is widely distributed in the soil is indicated by the report that fluorine ranks twentieth among the elements in frequency of occurrence.

For convenience in clarifying the various stages or severity of mottling of enamel as seen in man, H. T. Dean,[4] of the U. S. Public Health Service, prepared a set of drawings. (Fig. 3.) More recently the classifications "moderately severe" and "severe" have been grouped as "severe." (Dean, personal communication.)

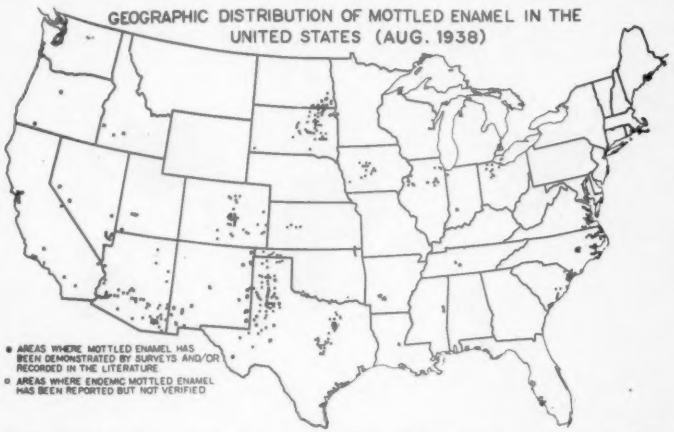
GEOGRAPHIC DISTRIBUTION OF MOTTLED ENAMEL IN THE UNITED STATES (AUG. 1938)
• AREAS WHERE MOTTLED ENAMEL HAS BEEN DEMONSTRATED BY SURVEYS AND/OR RECORDED IN THE LITERATURE
◦ AREAS WHERE ENDEMIC MOTTLED ENAMEL HAS BEEN REPORTED BUT NOT VERIFIED

Fig. 1.—Distribution of mottling of enamel in United States in August 1938. (From American Journal of Public Health.)

The relatively short life span of the albino rat, and the ease with which a characteristic enamel defect can be induced by administration of fluorine, make the albino rat the animal of choice for experimental fluorine poisoning. Since the enamel defect produced in the rat differs somewhat from that in man, it will be described briefly. The incisor teeth of the young albino rat are white when they erupt. Shortly thereafter, the enamel on the anterior surfaces of the upper and lower incisors develops a brown tint, which rapidly darkens until almost complete coloration is recorded between 20 and 30 days of age. The upper incisors are somewhat darker than the more rapidly growing lower incisors. If a fluorine compound, such as sodium fluoride, is mixed with the diet and fed to albino rats from the time of weaning, the coloration of the incisor teeth is either retarded or completely prevented, depending on the dosage of fluorine. Under these conditions, we have seldom seen the development of striations in the teeth. However, if fluorine is administered to albino rats old enough to have normally colored incisor teeth, loss of color occurs, but not uniformly over the whole anterior surface of the incisors. Close inspection of the incisors shows bands of normal coloration separated by white bands. As the period of feeling is continued, the bands of coloration gradually disappear, until the whole tooth presents an even white appearance. Reproductions of normal and banded rat incisor teeth are shown in Figures 4 and 5, respectively.

If, instead of feeding the sodium

fluoride mixed with the daily ration, a single dose of sodium fluoride solution is injected, a single white band will appear subsequently. By repeating the injection at regular or irregular intervals, a series of regularly or irregularly spaced white bands may be produced on the incisors.

Under conditions permitting a continuous daily intake of fluorine, by the addition of fluoride to the diet or to the drinking water, it is quite generally agreed that a concentration of 0.0014 per cent added fluorine, corresponding to 14 parts per million parts of food, will cause a detectable degree of bleaching of the albino rat incisor. Therefore, the rat is less susceptible than children, in whom a concentration of 1 part fluorine per million parts of drinking water is effective. This conclusion does not take into consideration the species difference in time required for calcification. However, the conclusion is supported by the report of Cox, Matuschak, Dixon and Walker,[5] who state, "The high degree of fluorine required to produce mottled molars is more evidence that the rat is less sensitive to fluorosis than man."

Another important difference between the rat and human species is related to the fact that, in rats, the incisor teeth grow continuously throughout life. In the rat, bleaching of the incisor teeth can be produced at any age by an adequate dosage of fluorine. In man, mottling is produced only in those teeth which were being formed at the time of the fluorine intake. Therefore, it is a defect limited to those individuals who used a water containing toxic amounts of fluorides during the susceptible period.

In the rat, chronic feeding of fluorine has been shown to cause retardation of growth rate. Figure 6 shows two litter mates, one of which received a normal (control) diet, while the other received a diet containing 0.1 per cent sodium fluoride. When growth is used as a criterion of toxicity, it may be easily demonstrated that the more soluble fluorine compounds, such as sodium fluoride, are more toxic than the less soluble compounds, such as cryolite (sodium aluminum fluoride). The dosage of fluorine required to cause bleaching of the rat incisors is so low that, when it is used as a criterion of toxicity, differences in water solubility of fluorine compounds become of little or no importance.

Fig. 2.—Countries or sections thereof in which occurrence of mottling of enamel in at least one area has been reported.

Another effect of fluorine that may be observed in rats is a loosening of the incisors in the sockets and a marked overgrowth, due to a loss of apposition. Such overgrowth, produced on a diet containing 0.05 per cent cryolite, is shown in

Figure 7. Figure 8 shows an upper incisor removed at necropsy.

Nature of Fluorine Action

The evidence concerning the mechanism by which fluorine induces its characteristic effect on teeth may now be summarized. This evidence is entirely concerned with the experimental rat. The action is not due to calcium deprivation, nor can it be counteracted by calcium-administration as is the case with acute fluorine poisoning. There is no change in blood calcium levels associated with feeding of fluorine in amounts capable of causing bleaching. These conclusions have been strikingly confirmed by experiments involving the feeding of diets to which dicalcium phosphate contaminated with fluorine had been added. On the basis of the known fluorine content, it was possible to add the dicalcium phosphate to the diet in such amounts that the added fluorine content corresponded to an amount known to be effective in bleaching rat incisors. Although large amounts of both calcium and phosphate were obviously added, in excess of the fluorine, bleaching occurred as readily as with an equivalent amount of fluorine in the form of sodium fluoride.

At necropsy, the ribs of fluorine-poisoned rats are seen to be whiter, thinner and more fragile than those of control rats. Alteration in the jaw bones caused by chronic fluorine poisoning has been the subject of investigation by Pachaly.[6] By vital staining with alizarin, he demonstrated the interference with calcium deposition in the maxillary bone.

Biochemical evidence obtained by us has given information with regard both to bone injury from chronic fluorine poisoning and to the mechanism of the injury. The work of Robison[7] has defi-

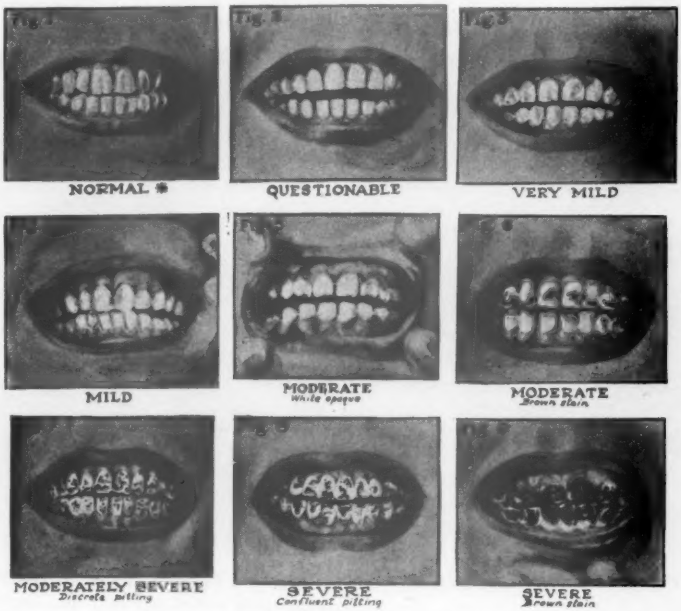

Fig. 3.—Degrees of mottling of enamel in man.

nitely established the significance of phosphoric esters in metabolism and the importance of the enzyme phosphatase in hydrolyzing these esters, the liberated phosphate ion being utilized in the process of calcification. Because of the action of fluorine compounds on enzymes in general, it seemed logical to investigate the action of fluorine on the enzyme phosphatase.

It has been claimed by Phillips[8] that chronic fluorine poisoning of heifers and swine is characterized by an increase in plasma phosphatase. Attempts by us to study plasma phosphatase in rats failed to yield any conclusive results. Therefore, attention was turned to a study of bone phosphatase.

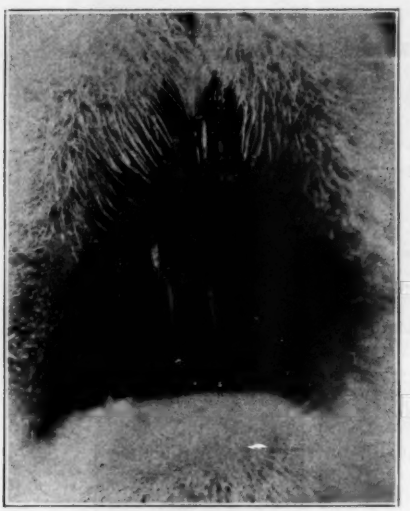

Fig. 4.—Normal rat incisors.

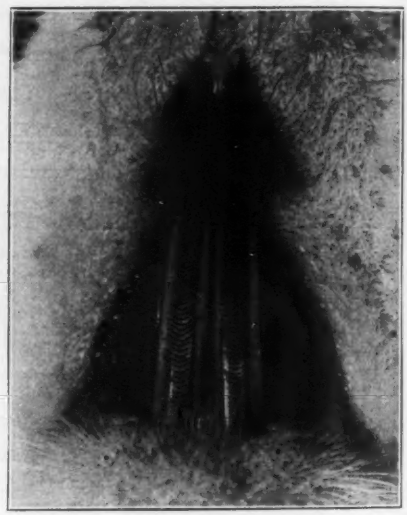

Fig. 5.—Banding of rat incisors from diet containing 0.0028 per cent fluorine.

When the bone phosphatase of fluorine-fed rats was compared with that of control rats, it was found that fluorine toxicity was associated with a decrease in bone phosphatase activity up to age limits of about 30 days. This relationship exists only under strictly controlled conditions.

The control and experimental rats must be obtained from litters of the same size and within one or two days of the same age. Two rats having litters within one or two days of each other were selected, and the larger litter was reduced in number to that of the smaller. Within one day after delivery of the litters, one of the mother rats was placed on a diet containing fluorine, the other being allowed to remain on the normal (control) diet. Up to the time of weaning, the only source of fluorine for the young rats in the experimental group was the mother's milk.

At five-day intervals, one control rat and one experimental rat were killed, and, after muscle and tendon tissues were carefully removed, determinations of bone phosphatase activity were made on the combined femur, fibula and tibia. Figure 9 illustrates the correlation between bone phosphatase activity and fluorine poisoning.

In addition to this correlation, it was found that the extent to which the bone phosphatase activity of the experimental

rats deviated from that of the controls agreed roughly with the deviation of the growth curve from the normal. The more nearly the experimental growth curve approximated a normal growth curve, the slighter the deviation in bone phosphatase activity.

To what extent can these results obtained *in vivo* be duplicated *in vitro?* To answer this question, and to obtain further information on the mechanism of fluorine poisoning, the ability of an active bone phosphatase preparation to hydrolyze sodium glycerophosphate in the presence of varying molar concentrations of sodium fluoride was studied. It was found that sodium fluoride decreased the activity of bone phosphatase and that the inhibition was roughly proportional to the fluoride concentration and could be demonstrated in a concentration as low as one two-hundredth molar.

The ingestion of cadmium has an effect on rat incisors indistinguishable from that of fluorine ingestion. Experiments with cadmium chloride *in vitro,* like those with fluorine, showed that cadmium chloride in a concentration of 10^{-3}N reduced the activity of bone phosphate to 8 per cent of that of controls. The fact that cadmium causes bleaching of rat incisors certainly militates against the idea that calcium deficiency plays a rôle in the action of fluorine.

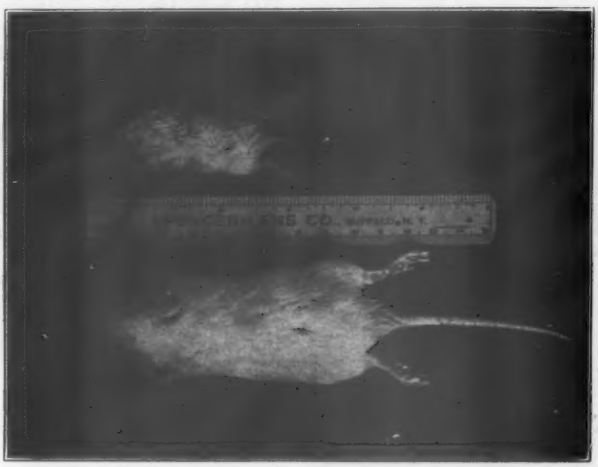

Fig. 6.—Litter mates. Inhibition of growth resulted in the animal above from a diet containing 0.1 per cent sodium fluoride.

ENDOCRINES AS FACTORS

In experimental fluorine poisoning, both acute and chronic, one finds a variation in susceptibility to dosage. Not all rats on a given daily intake respond with the same degree of bleaching of the incisor teeth. This is not surprising, for the data on most toxic agents reveal both a species and an individual variation in susceptibility. No doubt children born and reared in an endemic area and using the same water supply exhibit considerable variation in severity of mottling of the enamel. There are no doubt many factors, both intrinsic and extrinsic, in such variability. Usually, when pressed for an explanation of such variability, we say that it is due to a difference in functional state, but this is merely a restatement of the problem and adds nothing to the fund of knowledge. The

mechanism of toxic action varies from one toxic agent to another, and the factors that account for the variation in response or difference in functional state may account also for the results observed with one agent, but not with another. Considerable time might be spent in discussing the general aspects of this problem. Attention is called to the general problem of variability of response merely to emphasize the importance of the following observations.

In our studies on fluorine toxicity, we have observed two factors in producing a given functional state; i. e., in determining the response to a given daily intake of fluorine. These two factors are the activity of the thyroid gland and the secretion of thyrotropic hormone by the anterior pituitary gland. These merit discussion because of their probable practical importance in the problem of mottled enamel in man.

Fig. 7.—Loss of apposition and overgrowth of incisors from diet containing 0.05 per cent cryolite.

The literature on fluorine poisoning contains references to the relationship of both the thyroid and the pituitary glands to fluorine, but the interest has always centered on the effect of fluorine on these structures. No reference has been found on the reverse relationship, the effect of glandular activity on the toxicity of fluorine as manifested in the production of enamel dystrophy. The nearest approach to such a relationship was the observations of Phillips and his co-workers[9] and of Karnofsky and Cronkite.[10] The former authors showed that thyroid and fluorine exerted a synergistic effect on the basal metabolic rate. A given dose of fluorine was shown to have no effect on the basal metabolic rate, but when it was given in conjunction with thyroid, the effect was much greater than that from thyroid alone. The latter investigators demonstrated that the injection of thyroxin caused the teeth of young rats to erupt at an earlier date. Since rat teeth are normally white at the time of eruption, it is not surprising that no report was made on the effect of thyroxin on tooth coloration.

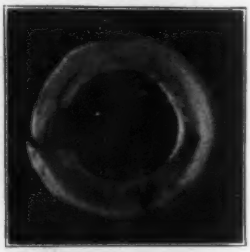

Fig. 8.—Marked overgrowth of upper incisor resulting from diet containing 0.05 per cent cryolite.

During the course of our work, it became necessary to induce overeating in albino rats, using a diet containing a small but known amount of fluorine. The details of this investigation are reported elsewhere,[11] but the salient features will be given here. Overeating may be induced by adding 0.2 per cent powdered thyroid to the basic rat diet employed, or by the daily injection of protamine zinc insulin. Since the in-

crease in food intake induced by the treatment with protamine zinc insulin was equal to or greater than that induced by feeding thyroid, and since the insulin treated rats exhibited no enhanced bleaching, it is evident that the greater daily fluorine intake resulting from an elevated food intake was not responsible for the marked bleaching seen in the thyroid fed rats. In brief, the feeding of powdered thyroid, equivalent to a state of hyperthyroidism, had a marked synergistic effect on the action of fluorine on tooth coloration. The results of this investigation are shown graphically in Figure 10.

In Figure 10, +++ represents complete bleaching of the incisor teeth; − indicates the brown color seen on the

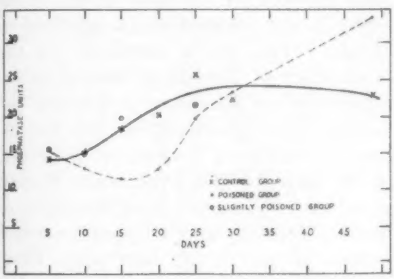

Fig. 9.—Correlation between bone phosphatase activity and fluorine poisoning.

tooth of the normal adult albino rat, and ++, + and ± correspond to intermediate degrees of bleaching. This arbitrary evaluation of the degree of bleaching permitted the observer to duplicate closely his estimate of the amount of bleaching even when the animal which was being examined was unknown, and the estimate of one observer would agree well with that of another. For purposes of plotting, the plusses assigned to the individual animals in each group were averaged.

A logical explanation for this synergistic action of thyroid on fluorine toxicity may readily be based on two observations. The explanation is related to the growth stimulation resulting from thyroid therapy. The first observation concerns the greater toxic action of fluorine on the lower incisor than on the upper. In normal rats and rabbits, the lower incisors grow more rapidly than do the upper, the latter growing about two-thirds the rate of the former. It is a repeatedly confirmed generalization that young or rapidly growing animals or tissues respond more readily to the toxic action of a variety of agents than do mature animals. In rats, fluorine bleaching occurs earlier or on a lower dosage on the rapidly growing lower incisors than on the slower growing upper incisors. The second observation is that

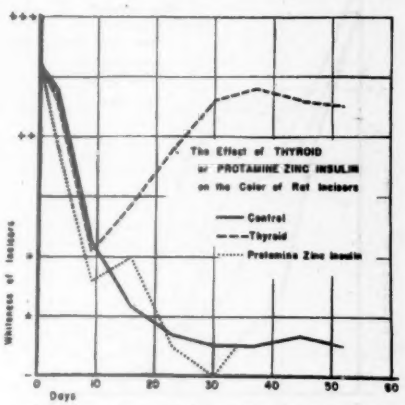

Fig. 10.—Synergistic action of thyroid on fluorine toxicity.

of Karnofsky and Cronkite,[10] who reported that the rate of incisor tooth eruption in rats is markedly accelerated by the injection of thyroxin. These two observations suggest that the toxic action of a given dosage of fluorine is enhanced by the increased rate of differentiation and growth of the organism caused by feeding thyroid.

In view of this synergistic action of thyroid on fluorine toxicity, it may be expected that the thyrotropic hormone secreted by the anterior pituitary might also sensitize one to the toxic action of

fluorine, an action mediated through the stimulation of the thyroid gland. Since the details of the investigation designed to test this possibility have been reported elsewhere,[12] only the results need be summarized here.

Four groups of rats approximately the same age and weight were used. The basic diet given to all rats contained 0.001 per cent fluorine, an amount too small to seriously retard the rate of tooth coloration in the growing rat. One group served as controls and the three other groups received daily except Sunday subcutaneous injections of 0.02, 0.1 and 0.5 cc. of thyrotropic hormone prepared from beef anterior pituitary glands by the flavianate method.[13] The rate of incisor tooth coloration was evaluated and plotted graphically as previously described. The results of the experiment are shown graphically in Figure 11.

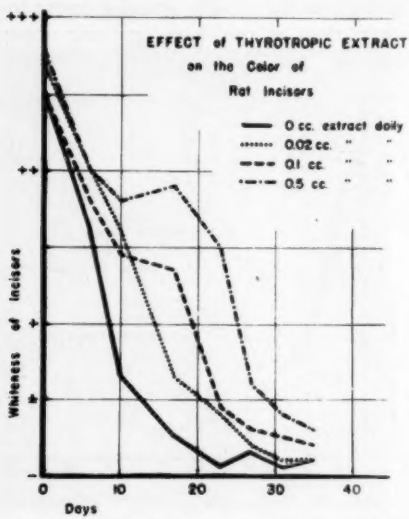

Fig. 11.—Effect of thyrotropic hormone ingestion on fluorine toxicity.

The graph shows that the characteristic coloration of the incisor teeth of the control rats proceeded normally. The lowest dosage of thyrotropic hormone permitted rapid tooth coloration, but at a rate somewhat slower than in the control group. The rats receiving 0.1 cc. of the hormone showed an almost normal rate of coloration until the tenth day, but, from the tenth to the seventeenth day, the rate of coloration was definitely retarded. On the highest dosage, 0.5 cc. of thyrotropic hormone, the rate of tooth coloration started to deviate sharply from normal on the tenth day, and actually showed increased bleaching until the seventeenth day, after which the development of incisor tooth coloration was resumed. Since rats become refractory to the action of thyrotropic hormone, the bleaching effect of fluorine does not persist.

Thus far, experimental evidence has been summarized on three points of interest in the production of mottling of enamel. The points concern (1) the mechanism of fluorine poisoning as exhibited in an inhibitory action on the enzyme bone phosphatase, (2) the increased sensitivity to fluorine poisoning in the presence of a hyperactive thyroid gland and (3) an increased sensitivity to fluorine brought about by an increased output of thyrotropic hormone. These last two points are believed to be important factors in the individual variation in susceptibility to fluorine poisoning.

One other point of interest to the dental profession should be mentioned. Reference is made to the recent evidence[14] that ingestion of fluorine lowers the incidence of dental caries. Fluorine occurs naturally in the mineral apatite in combination with phosphate. It is interesting that Gautier and Clausmann[15] found that the fluorine of the tissues is localized in a specific manner, and that fluorine accompanies the alkaline earth phosphates and increases with them. A natural characteristic of calcium fluorphosphate is hardness, or resistance to abrasion. If, as suggested by the report of Gautier and

Clausmann, the fluorine found in teeth is in the form of fluorapatite, it could modify the hardness of teeth and conceivably make them more resistant to caries.

CADMIUM

Earlier in this discussion, mention was made of the fact that cadmium ingestion has an effect on rat incisors indistinguishable from that of fluorine. The details of the investigation on the toxic action of cadmium will be published elsewhere, but the phase of the work relating to mottled enamel may be discussed here because of its theoretical interest and possible practical importance to dentists.

Does cadmium sensitize or predispose the animal to the effect of subminimal amounts of fluorine unavoidably present in the diet? In other words, are the effects of cadmium and fluorine ingestion synergistic or additive? Since we were not successful in designing experiments to answer these questions conclusively, only one of the experiments demonstrating the action of cadmium will be described.

The most direct method of answering these questions would be to compare a control group of animals on a basic diet known to be free of fluorine with a group of rats on the same fluorine-free diet, but containing cadmium. If bleaching of the

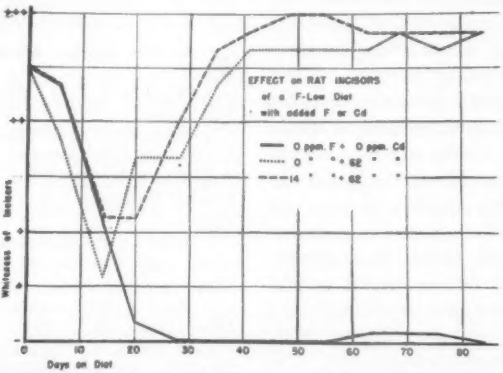

Fig. 12.—Bleaching of rat incisors from diet containing 0.0062 per cent cadmium.

The studies on the chronic toxicity of orally ingested cadmium demonstrated that the most delicate criterion of toxicity in the albino rat was bleaching of the incisors similar to, if not identical with, that associated with fluorine. On a diet containing 0.0016 per cent of added cadmium in the form of cadmium chloride, albino rats grew normally, but exhibited an unmistakable degree of bleaching of the incisor teeth. This unexpected and striking observation raises several questions. Is the enamel defect caused by the ingestion of cadmium the same as that caused by fluorine? Is cadmium *per se* responsible for the effect? incisors resulted in the latter group, cadmium would be shown to be the causative agent. If there was no bleaching in the absence of fluorine, the cadmium action would have to be regarded as due to a sensitization to fluorine. Unfortunately, the preparation of an absolutely fluorine-free diet has not yet been accomplished. Since it seemed reasonable to assume that the bone ash used in the preparation of the basic diet was the principal source of the native fluorine, a basic diet was prepared with the following modification: The bone ash was omitted, but approximately the same concentrations of calcium, magnesium

and phosphorus were attained by the addition of c.p. chemicals.

Three groups of three rats each were used, as follows: The first group served as controls and received the supposedly low-fluorine basic diet. The second group received the same diet plus 0.0062 per cent cadmium. The third group received the basic diet plus 0.0062 per cent cadmium and 0.0014 per cent fluorine. Coloration of the incisor teeth was evaluated and plotted graphically as described above. (Fig. 12.)

It will be seen that the curve for the control group of rats drops abruptly to the base line and remains close thereto, thus indicating a low native fluorine content in the basic diet. The other two curves show that coloration of the teeth started to develop, but in about fourteen days a bleaching effect became evident and proceeded rapidly thereafter. In view of the low native fluorine content of the basic diet, these results suggest, but do not prove conclusively, that cadmium ingestion *per se* can cause bleaching of rat incisors and that the action is not due to a sensitization of the rats to fluorine present in the diet. This conclusion is supported by the fact that cadmium is a potent inhibitor of the enzyme bone phosphatase *in vitro*.

Since the enamel defect produced in rats by fluorine ingestion is generally admitted to be the counterpart of that produced in man, and since cadmium ingestion produces the same or similar action in rats, it may be concluded that cadmium can cause enamel dystrophy in man. The incidence of exposure to cadmium being unknown, it is not possible to assert that any of the cases of mottled enamel are due to chronic ingestion of cadmium. Perhaps the chief value of this report on the toxicity of cadmium lies in its prophetic nature.

SUMMARY

1. In the experimental production of defective enamel in rats, the toxic action of fluorine may be associated with its inhibitory effect on the enzyme phosphatase.

2. Administration of thyroid greatly enhances the susceptibility to fluorine poisoning.

3. Administration of the thyrotropic hormone secreted by the anterior pituitary gland increases susceptibility to fluorine toxicity.

4. Variations in the activity of the thyroid and pituitary glands may be factors in the individual variation in susceptibility to fluorine toxicity.

5. Cadmium ingestion causes defects of the enamel in rats and in man.

BIBLIOGRAPHY

1. DEEDS, FLOYD: *Medicine*, 12:1, February 1933.

2. MCCLURE, F. J.: *Physiol. Rev.*, 13:277, July 1933.

3. ROHOLM, K.: Fluorine Intoxication. London: H. K. Lewis and Co., Ltd., 1937.

4. DEAN, H. T.: Classification of Mottled Enamel Diagnosis. J.A.D.A., 21:1421, August 1934.

5. COX, G. J.; MATUSCHAK, M. C.; DIXON, S. F., and WALKER, W. E.: *Science*, 90:83, 1939.

6. PACHALY, W.: *Arch. f. exper. Path. u. Pharmacol.*, 166:1, 1932.

7. ROBISON, R.: Significance of Phosphoric Esters in Metabolism. New York: New York University Press, 1932.

8. PHILLIPS, P. H.: *Science*, 76:239, 1932.

9. PHILLIPS, P. H.; ENGLISH, H. E., and HART, E. B.: *Am. J. Physiol.*, 113:441, October 1935.

10. KARNOFSKY, D., and CRONKITE, E. P.: *Proc. Soc. Exper. Biol. & Med.*, 40:568, April 1939.

11. WILSON, R. H., and DEEDS, FLOYD: *Endocrinology*. 26:851, May 1940.

12. DEEDS, FLOYD; WILSON, R. H., and CUTTING, W. G.: *Endocrinology*, 26:1053, June 1940.

13. EVANS, H. M.; MEYER, K., and SIMPSON, M. E.: *Mem. Univ. Calif.*, 11:27, 1933.

14. DEAN, H. T.; JAY, P.; ARNOLD, F. A., JR.; MCCLURE, F. J., and ELVOVE, E.: *Pub. Health Rep.*, 54:862, May 26, 1939.

15. GAUTIER, A., and CLAUSMANN, P.: *Compt. rend. Acad. d. Sc.*, 156:94 and 1425, 1913.

THE IMPACTED MANDIBULAR THIRD MOLAR: CLASSIFICATION, DIAGNOSIS AND OPERATIVE TECHNIC

By RALPH B. RODE, D.D.S., St. Louis, Mo.

TODAY, the profession of dentistry is not only a recognized science of the first order: it boasts of a growth and development equal in rapidity and extension to that of any allied medical or surgical science. Yet, it is just a century ago that the first known dental school was established in Baltimore, Md. Prior to that time, there was little literature on the subject.

Probably one of the most remarkable features of this incredible development is oral surgery, a subject which progressed but slowly during the early years after its inception, but a practice that, in our own time, has attained an importance equal in dentistry to general surgery in medicine.

The man who is considered the "father" of this comparatively new science was James Edward Garretson, who undoubtedly received much of his inspiration from the work of his brilliant predecessor, Simon P. Hullihen. Garretson was a graduate dentist and professor of anatomy in the Philadelphia School of Anatomy from 1862 to 1867. At that time, oral surgery was the province of either the general surgeon or the dentist. There was no legislation regulating the practice of the respective professions. It was during his tenure as professor of anatomy that Garretson became convinced that this branch of surgery called for special training. In 1869, although there was much opposition to specialties on the part of the medical profession, Garretson was appointed oral surgeon to the hospital of the University of Pennsylvania, the first official recognition and the birthday of oral surgery as a specialty.

In 1895, William Konrad Roentgen announced the discovery of the roentgen rays, the diagnostic value of which dentistry recognized almost immediately. In 1896, only one year after its discovery, C. Edmund Kells, a dentist, of New Orleans, gave the first dental roentgen ray clinic before the Southern Dental Association.

The development of roentgenology is perhaps the most powerful stimulus that dentistry has ever received; for certainly it has benefited every phase of dentistry. Through its use, we are able to identify conditions which would not be apparent from clinical examination and to avoid guesswork in diagnosis, thus building technic on a solid foundation of fact. But what is perhaps most significant and encouraging is the fact that roentgenology revealed pathologic conditions which have been found to be contributing factors in systemic disturbance, and thereby brought medicine and dentistry into a working partnership.

Roentgenology permitted a standardization of technic in oral surgery that greatly advanced the progress of the specialty. The World War provided

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further impetus in that many dentists who observed and had experience in oral surgery during the war became interested in the subject and took up the specialty on their return to private practice.

It was during those progressive years just prior to the World War that George B. Winter began, with the help of roentgenology, to evolve his classification and diagnosis of impactions of the mandibular third molar and to base his operative technic on that classification and diagnosis. The nomenclature he gave to the classification is as follows: vertical, mesioangular, distoangular and horizontal impaction, with variations in deflections and anomalies.

This classification required several years of intensive research. Old burying grounds were searched for mandibles containing impacted third molars. Cross-sections were made of some of these mandibles and they were thoroughly studied with the aid of roentgenology and photography. Wires were placed in the inferior dental canal to determine the relation of the canal to the roots of the mandibular third molar. Extracted mandibular third molars were examined and photographed to determine the various sizes and shapes of the crowns, the root formations and the anatomic variations. Impression study models were made of the third molar region in order to note the relation of the supporting structures.

Before any progress could be made in the classification, there had to be a standardized roentgenologic technic for the impacted mandibular third molar. The film is placed on the lingual side of the teeth with the side of the film at a right angle to the target rays. The patient's forefinger is used to hold the film in place so that it is firm against the teeth but does not exert enough pressure to bend the film buccally. The occlusal surfaces of the molar teeth must be on a horizontal plane. Where the crowns of the molar teeth are tilted lingually to some extent, it is necessary to tilt the patient's head in the opposite direction to keep the occlusal surfaces on a horizontal plane. For a vertical impaction, the proximal end of the film protector is at the mesial surface of the first molar. When the third molar is impacted in a mesioangular, distoangular or horizontal position, the proximal end of the film protector is at the center of the first molar.

In addition to the lingual view roentgenogram, an occlusal view is often necessary for a complete diagnosis of an impaction of the mandibular third molar. The film is placed on the occlusal surface of the teeth with the proximal end of the film protector at the center of the first molar and in such manner that there is an equal amount of film on each side, buccal and lingual, of the molar teeth. The patient is asked to close the mouth, biting to hold the film in place. Originally, the patient held the film in place with the forefinger, but it was found that closing the mouth relaxed the soft tissues and let the chin come forward for better angulation. The head is tilted so that the side of the mandible containing the impacted tooth is turned upward and elevated as far as possible. The cone is directed upward at an obtuse angle as closely as the case will permit toward the inferior border of the mandible. The cone is then adjusted so that the rays will pass in a straight line through the inferior border of the mandible to the crown of the impacted third molar and will cast a perfect shadow of the crown upon the roentgenogram, but will not show any part of the roots of the first and second molars, as they will be superimposed upon the crowns if the roentgenogram is correctly made.

An extra-oral film is necessary when an odontoma, trismus, cellulitis or a cystic formation is present, or when the

third molar is in an unusual position.

The importance of a correctly made roentgenogram cannot be overemphasized. If the film is correctly made, with the normal third molar as a control, any deviation from the normal can be immediately detected. Any degree of distortion due to incorrect angulation may lead to a false diagnostic conclusion, which will complicate operative procedure.

After a true picture of an impacted mandibular third molar has been obtained and supplemented by clinical examination, there are a number of general routine observations to be made. These are as follows: the condition of the enamel cap of the first and second molars; the interproximal contact of the first and second molars; the relation of the third molar to the occlusal surfaces of the first and second molars; the condition of the pulp chambers of the first, second and third molars, and of the crown of the second molar; deflections; the condition of the inferior dental canal, and evidences of pathologic involvement.

From a lingual view roentgenogram, the diagnostic points that govern the instrumentation and operative technic are as follows: (1) the distance for access; (2) the contact of the crown of the third molar with the second molar; (3) accessibility or inaccessibility; (4) the height of the superior border of the ossistructure; (5) the condition of the interseptum; (6) the size and shape of the mesial root; (7) the condition of the septum; (8) the size and shape of the distal root; (9) the size and shape of the crown; (10) the distal ossistructure.

When an impacted mandibular third molar is said to be accessible, no excision of ossistructure is necessary in order to apply the exolever at the mesiobuccal surface. When it is inaccessible, excision of ossistructure is necessary in order to apply the exolever at the mesiobuccal surface.

The impacted mandibular third molar may be in alinement with the first and second molars, in which case it is termed a no deflection. It may be out of alinement buccally and is termed a buccal deflection; out of alinement lingually and is termed a lingual deflection; out of alinement buccolingually and is termed a buccolingual deflection. The degree of deflection is determined from the diagnostic points shown in the occlusal view roentgenogram as compared with the lingual view and clinical examination.

The occlusal view also establishes the available fulcrum for leverage. It shows the ossistructure lingually from the impacted tooth and definitely eliminates it for use as a fulcrum, owing to its being thin and easily fractured. It allows the determination of any ledges of the lingual or buccal plates that may extend over a part of the occlusal surface. It shows any space along the buccal side or such a v-shaped space as sometimes exists at the mesiobuccal surface. It reveals any pathologic condition existing along the buccal side, which, if present, will greatly reduce the amount of excision when operative procedure is instituted.

The comparative alinements to be determined from the lingual and occlusal view roentgenograms are as follows: the alinement of the buccal and lingual surfaces of the third molar when there is no deflection with the corresponding surfaces of the second molar; the alinement of the third molar when a part of the crown is deflected buccally out of alinement with the distobuccal surface of the second molar; the alinement of the third molar when its occlusal and buccal surfaces are deflected to a slight extent lingually, and the alinement of the third molar when its occlusal surface is deflected buccolingually.

In all classifications of the mandibular

third molar when impacted, the degree of impaction may vary in cases having the same deflection. With the vertical impaction as an illustration, in a number of cases presenting no deflection, one may be a case in which the superior border of the ossistructure extends up to the gingival third, leaving the buccal surface free of ossistructure, and another may be a case where the superior border of the ossistructure extends up to the occlusal surface, indicating that the ossistructure extends over the entire buccal surface. In an occasional case, the ossistructure may extend over the entire distal, occlusal and buccal surfaces of the third molar.

In a mesioangular impaction of the third molar, the same deflections are found as in the vertical, but the degree of deflection may be more extensive. In the distoangular impaction, the deflections are not so marked as in the vertical and mesioangular types. In the horizontal impaction, the deflections are more pronounced than in any other type.

Occasionally, an extreme lingual deflection of a horizontal impaction is encountered, with the tooth turned completely crosswise, when it is classified as a complete lingual impaction. A lingual view roentgenogram will show only the occlusal surface of the crown, with no delineation whatever of the root formation. In order to obtain even an approximate idea of the root formation, a distorted lingual view is made in addition to the occlusal view. The film is placed in the usual manner, but the cone of the machine is tilted as in an exposure for an upper molar. This angulation will cause an elongation of the first and second molars, but will bring out the root formation of the third molar to some extent.

A torsional deflection may be recognized in a lingual view roentgenogram by making a comparison of the pulp chambers and pulp canals of the third molar with those of the first and second molars.

The inverted type of impacted mandibular third molar is an unusual rather than a standard classification.

The root formation to a large extent governs the amount of ossistructure that must be excised and the lever pressure that may be exerted. The root classification is as follows: mesial root distal and distal root straight; roots inclined distally; roots fused; both roots straight and separated; mesial root distal, distal root mesial; mesial root straight, distal root mesial; extra root; divergent roots; hypercementosed roots; contacting of roots.

Before operative technic is considered, the ten diagnostic points on which it is based bear repetition. They are as follows: (1) distance for access; (2) contact of the crown of the third molar with the second molar; (3) accessibility or inaccessibility; (4) height of the superior border of the ossistructure; (5) the condition of the interseptum; (6) size and shape of the mesial root; (7) condition of the septum; (8) size and shape of the distal root; (9) size and shape of the crown; (10) distal ossistructure.

The various deflections that may be encountered must be considered, as no deflection, buccal deflection, lingual deflection, buccolingual deflection and torsional deflection, all with the mesial surface either accessible or inaccessible.

In a vertical impaction, no deflection, mesial surface accessible, after incision of the gum tissue has been made, excision of the ossistructure distally from the third molar is made to permit the tooth to be directed distally and upward, the extent of the excision being governed by the root formation. The blade of the ossisector is directed distally to the part to be excised at a slight angle toward the tooth, always excising toward the tooth. Where the ossistructure extends to the distolingual surface or over the disto-

clusal surface of the third molar, repeated applications of the ossisector may be necessary in order to excise all of the ossistructure over the involved surface.

Examination of the ossistructure on the buccal surface must then be made to determine whether the exolever can be applied to the bifurcation at the buccal surface in order to direct the tooth distally and upward in line with its long axis. If access cannot be obtained to the bifurcation, a half-moon excision of the buccal ossistructure is made at that point. The osseous tissue should be left smooth and rounded and excision should be only sufficient to permit application of the exolever, as the heavy cortical plate is to be used as a fulcrum.

After this excision has been made, an exolever with a blade of suitable size is applied to the mesiobuccal surface to direct the tooth upward and distally as far as the character of the root formation will permit. An exolever is then applied to the buccal surface at the bifurcation to complete the extraction.

In the vertical impaction, where the mesial surface is inaccessible, after sufficient excision has been made distally from the third molar, the blade of an ossisector is directed downward on the mesiobuccal surface, excising sufficient ossistructure to create a space for the application of an exolever to the mesiobuccal surface. If there is a contact of the mesial root of the third molar with the distal root of the second molar, and the third molar cannot be directed distally on account of the liability of fracture of the distal root of the second molar, an exolever is applied at the mesiobuccal surface to create a wedge and pressure is exerted downward in order to direct the tooth upward out of the socket in line with its long axis.

In a mesioangular impaction, no deflection, mesial surface accessible, when the ossistructure is below or to the distal surface of the third molar, sufficient excision is made distally from the third molar to allow the tooth to be directed distally and upward, in conformity with the root formation, into the space created by the distal excision. The size of the exolever blade which is applied to the mesiobuccal surface in order to direct the tooth distally and upward is governed by the amount of space on this surface which is shown in the lingual view roentgenogram. After the tooth has been directed distally and upward to a point where the mesioclusal surface of the third molar will clear the distal surface of the second molar, a buccal application of the exolever is made to complete the extraction.

In a mesioangular impaction with the mesial surface inaccessible, after the distal excision has been made, it will be necessary to make sufficient excision on the mesiobuccal surface for the application of the exolever to this surface. Where the ossistructure extends partially or completely over the occlusal surface of the third molar, it must be removed. Excision on the occlusal surface is indicated in advance of the distal and mesiobuccal excision of ossistructure. The blade of the ossisector is directed toward the occlusal surface of the third molar and excision continued until the entire surface is free. After the occlusal excision has been made, distal and mesiobuccal excision is made as previously described, followed by a mesiobuccal application of the exolever to complete the extraction.

The mandibular third molar, when impacted in a distoangular position, presents more complications than are found in the other standard classifications. In advance of operative procedure, the contact of the apical third of the root of the third molar with the distal root of the second must be noted. As a rule, no part of the crown is exposed but, if any part is exposed, it will be the mesial surface.

The excision of ossistructure on the distal, lingual and buccal surfaces will be more extensive in order to permit the tooth to be directed upward in line with its long axis, as any distal direction of the third molar might cause a fracture of the distal root of the second molar. In this type of impaction, it is essential that any ossistructure impinging on the occlusal surface be removed before application of an exolever. Again, an exolever is applied to create a wedge between the mesiobuccal ossistructure and the mesiobuccal surface of the third molar, exerting pressure downward in order to direct the tooth upward out of the socket in line with its long axis.

When the third molar is impacted in a horizontal position, the occlusal surface of the crown is in a vertical position as compared with the corresponding surface of the second molar. As in all classifications, previous to operative procedure, the diagnostic points are determined from clinical examination and comparison of the lingual and occlusal view roentgenograms. The extent of the gum tissue incision is determined by the type of deflection. After the gum tissue is displaced with the back of the ossisector blade, excision of ossistructure is made distally from the third molar, directing the blade slightly distally from the part to be excised and excising toward the third molar. After necessary excision has been made, the exolever technic is as described for a mesioangular impaction.

If the ossistructure is completely over the third molar, an instrument with a pointed blade is used for the initial penetration, after which excision is continued distally from the third molar.

If the mesial surface is inaccessible, excision on the mesiobuccal surface is necessary in order to permit application of the exolever to the mesiobuccal surface.

When the third molar is impacted in a horizontal position and buccally deflected, the amount of cortical plate on the buccal surface and the application of the exolever must be carefully considered. When the third molar is buccally out of alinement with the first and second molars, the width of the cortical plate on the buccal surface is reduced in comparison with a case in which there is no deflection. The application of the exolever must be made to the buccoclusal surface instead of to the mesiobuccal surface. A lifting movement distally and upward is made with the exolever in this type of case, using as a fulcrum the cortical plate anterior to the occlusal surface of the third molar instead of the cortical plate on the buccal surface.

Where there is a buccolingual deflection, excision on the buccal and distal surfaces must be more extensive and all impinging ossistructure on the occlusal, distal and lingual surfaces must be removed, as, in this type of case, the lingual cortical plate is extremely thin and any undue pressure exerted in moving the tooth out of the socket may result in fracture of the lingual plate.

As is the case in any operative procedure, the technic of third molar extraction is subject to such modification and improvement as may be indicated by existing conditions and experiences. The prime object in any application of the technic that I have described is to eliminate explorative manipulation in advance of or during operative procedure and to reduce trauma to a minimum.

906 Olive Street.

MIGRATORY ABSCESS OF RIGHT MANDIBULAR AREA

REPORT OF CASE

By GUSTAV C. TASSMAN,* D.D.S., and L. S. BLUMBERG,† D.D.S., Philadelphia, Pa.

History.—A. C., a white woman, aged 33, married, first seen January 25, 1941, in the accident ward of the Jewish Hospital, reported moderate pain with some swelling in the right mandibular area of two days' duration.

The patient had had the lower right second and third molars extracted by her family dentist two weeks previously. She stated that there was no undue difficulty or complication at the time with the exception of being "stuck with a needle about eight times." Nor was there any abnormal postoperative discomfort. There seemed to be normal healing of the sockets with no evidence of alveolitis.

When new developments appeared about ten days after the operation, the patient returned to her family dentist, who assured her that the condition would resolve itself without intervention. When no relief was apparent, the patient consulted her family physician, who referred her to the Jewish Hospital.

Examination.—There was frank swelling externally in the right mandibular area just below the angle of the jaw, with no localization. Cervical lymphadenopathy was noted. There was almost complete trismus, the patient being able to open her mouth only a quarter of an inch. Digital and visual examination revealed little except some swelling in the buccal vestibule in the region of the extracted teeth. The pulse rate and temperature were found to be slightly elevated.

Lateral jaw and intraoral films revealed no injury or osteomyelitis of the right side of the mandible. The sockets from which the teeth were extracted appeared clear. A soft-tissue film failed to reveal a localized soft tissue mass.

Treatment.—Conservative treatment was employed: hot saline mouthwashes every hour; ice applied externally; sedatives in the form of acetyl salicylic acid 10 grains with codeine sulfate one-half grain as needed; and a diet of fruit juice, milk, broth, soup and thin cereal. The patient was instructed to return to the clinic in two days.

When the patient returned, there had been no alleviation of symptoms and she was very uncomfortable. X-ray therapy was ordered locally and 100 r with a 1 mm. aluminum filter was administered. Sedation was continued, but the heat and cold were discontinued. The patient was instructed to continue with the diet as ordered and to return in two days.

Second Examination and Treatment.—The next examination (two days after the x-ray therapy) revealed that the patient was asymptomatic, grossly. The trismus and swelling had both disappeared. On oral examination, a localized abscess was seen on the posterior portion of the soft palate to the right of the midline. The area was incised without anesthesia. A thick creamy yellowish pus welled through the incision. The incision was enlarged with a mosquito hemostat to permit more complete drainage. A laboratory examination revealed gram-positive cocci (smear) and *Micrococcus catarrhalis* and pneumococci (culture). Hot saline mouthwashes were ordered to encourage drainage and further localization.

The following day, the patient returned. The incision was explored and more exudate was obtained. The area was irrigated with warm potassium permanganate. The area was painted with tincture of metaphen.

*Chief, Dental Staff.
†Dental intern.

An antiseptic mouth rinse was ordered to be used every three hours.

Two days later, the same treatment was given. At this time, it was noted that there was no longer any discharge from the area and that the wound was perfectly clean.

Outcome.—One week later, examination revealed complete absence of infection, with tissue regeneration normal. Final examination, February 19, 1941, revealed complete closure of the wound and the area practically healed. The patient was discharged as clinically cured. The final diagnosis was migratory abscess of the eighth mandibular area.

Medical Arts Building.
616 Spruce Street.

THE TWIN-TOOTH PONTIC IN THE CONSTRUCTION OF AN ALL-PORCELAIN BRIDGE

By A. H. TAMARIN, D.D.S., Chicago, Ill.

THE bridgework of today is beset with many difficulties and, even with the numerous advancements in technic, presents many problems. After many efforts on the part of the pioneers in bridgework, no pontic had been found that is completely satisfactory. Whenever a pontic brought strength and durability to the bridge, it was insanitary and of poor appearance, and vice versa.

Among the latest achievements in bridgework, only two types are of particular value:

The all-porcelain bridge which is baked on a platinum and iridium bar. This kind of bridge has the following characteristics:

1. It is 100 per cent esthetic.
2. It is 100 per cent sanitary.
3. It is too expensive for the average patient.
4. It is not practical. In the event that any one of its pontics breaks, the entire bridge must be removed. This, naturally, causes embarrassment to the dentist, as well as loss of time, prestige and money.
5. Its construction is difficult. The average dentist is not equipped, nor has he the time, to produce a bridge of that kind; hence, he is dependent on the laboratory.

The all-porcelain bridge made up of individual jacket crowns cemented on gold or platinum cores; which is:

1. One hundred per cent esthetic.
2. Practical. If one of the jacket crowns should break, it can be replaced without disturbing the rest of the bridge.
3. Not 100 per cent sanitary. The metallic core which rests upon the gum tissue cannot present so clean a surface as that of the all-porcelain bridge baked on a platinum bar.
4. Too expensive when made by the laboratory.
5. Difficult to construct. This kind of bridgework is quite complicated. The cores have to be molded, alined, cast and soldered to the abutments, and individual jacket crowns added to fit each individual core.

The kind of all-porcelain pontic (Fig. 1) that I am presenting to the profession possesses all the requirements for an ideal bridge. It will also, from an economical standpoint, replace all other

Clinic presented at the Seventy-Seventh Annual Midwinter Meeting of the Chicago Dental Society, February 17, 1941.

types of previous bridgework, and bring greater returns in fees to the dentist.

Let us now examine this pontic as to its appearance, sanitation, durability, economy and ease of construction.

1. Appearance:

This twin-tooth pontic is made up of two separate porcelain parts. (Figs. 2 and 3.) The first part (Fig. 2), which is of porcelain, includes the following

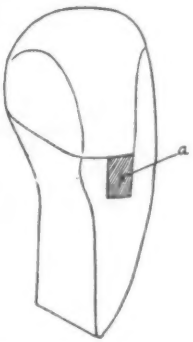

Fig. 1.—External appearance of twin-tooth pontic including rectangular surface of rod which forms its contact point.

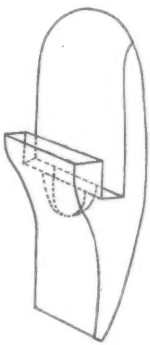

Fig. 2.—Part of twin-tooth which includes labial and lingual-incisal surfaces.

surfaces: the entire labial and incisal and part of the lingual surface of the incisors and cuspids, and the buccal, occlusal and parts of the lingual, mesial and distal surfaces of the molars and bicuspids. These porcelain surfaces give 100 per cent satisfaction as regards appearance, because no metal is visible. Also, the natural translucency of the incisal edges of the incisors and occlusal surfaces of the molars is maintained.

2. Sanitation:

The second part of this twin-tooth pontic (Fig. 3) is also all porcelain and covers the upper part of the lingual surface plus the entire base which rests

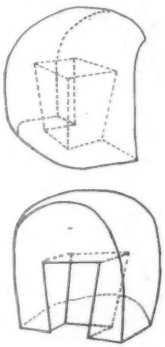

Fig. 3.—Part of twin-tooth which includes lingual-gingival surfaces.

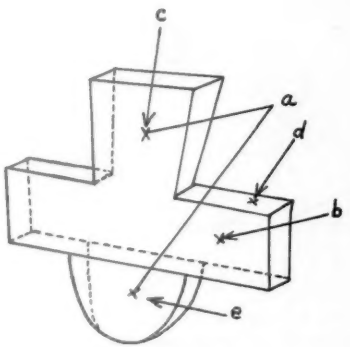

Fig. 4.—Metallic rod.

upon the gum tissue. This part of the pontic gives the twin-tooth 100 per cent sanitation. When these two parts of the twin-tooth are put together, they represent an all-porcelain tooth (Fig. 1) which has the appearance of a Goslee crown combined with the sanitation of

a "true pontic," or the appearance and sanitation of an all-porcelain bridge baked upon a platinum and iridium bar.

3. Durability:

Inside this twin-tooth, we have a metallic rod so constructed that it gives durability and strength to the first part of the twin-tooth and retention and strength to the second part of the twin-tooth. It also forms a strong union with its adjacent twin-tooth or abutments. The reinforcing metal rod (Fig. 4) is similar in design to a cross and does not necessarily have to be of a precious metal, since 100 per cent of the rod is embedded in porcelain and no part of it is ever exposed to the fluids of the mouth, nor is it in contact with the soft tissues of the mouth. The vertical or longitudinal part of this rod (Fig. 4, a) runs parallel to the long axis of the tooth. The upper portion of the vertical part of the rod gives retention to that part of the twin-tooth which comes in contact with the gum tissue. (Fig. 3.) The lower portion of the vertical part of the rod forms a tonguelike projection which gives stability and strength to the other part of the twin-tooth. (Fig. 2.)

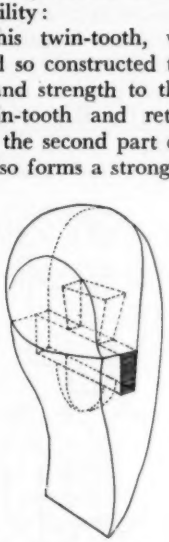

Fig. 5.—Position of metallic rod in relation to twin-tooth pontic.

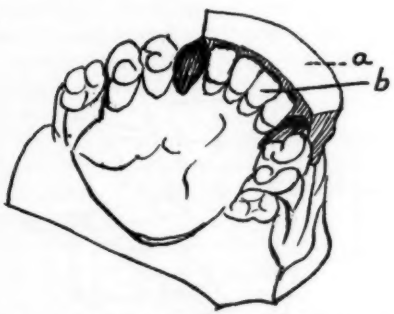

Fig. 6.—Cast of investment compound, with twin-teeth properly alined, supported by plaster form or core.

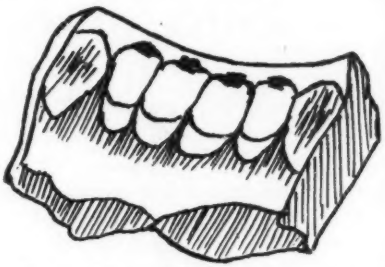

Fig. 7.—Plaster form with pontics in position, waxed together at incisal edges.

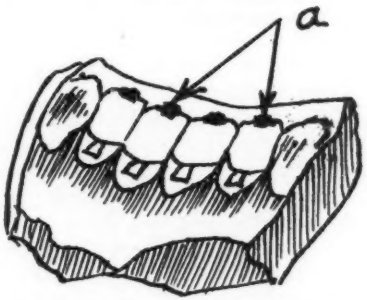

Fig. 8.—Pontics in position with lingual-gingival portions removed and upper parts of longitudinal portions of rods exposed.

The transverse portion of this rod (Fig. 4, b) forms two strong arms which terminate on the mesial and distal surfaces of the twin-tooth, presenting a rectangular appearance exactly where the contact points are supposed to be. (Fig. 1, a.) The entire transverse portion of the rod, besides the projecting tongue of the longitudinal part of the

rod, gives also support and strength to the lower portion of the twin-tooth. (Fig. 2.) After the rectangular surfaces (Fig. 1, a) of the adjacent rods are soldered together, they contact each other and also the abutments. They then form a continuous rod with the two abutments at each end of it. We then cement the two parts of the twin-tooth in position, and the entire bridge is ready for cementation in the mouth.

Fig. 9.—German silver wire attached to exposed surfaces of rods and abutments with sticky wax.

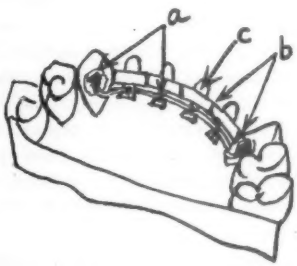

Fig. 10.—Rods held in position by german-silver wire, waxed up to upper parts of longitudinal sections.

4. Practicability:

The upper portion of the twin-tooth (Fig. 3) is anchored on and rests securely upon the upper portion of the longitudinal part of the rod (Fig. 4, c) and also upon the two shoulders of the transverse portion of the rod. (Fig. 4, d.) This upper portion of the twin-tooth has the greatest thickness of porcelain, and is also far away from the force of mastication. This part of the twin-tooth, in my estimation, can never break. Should an accident of that kind occur, it can easily be replaced by taking a modeling compound impression and running an amalgam die. We then select a similar portion and adjust it according to the amalgam die and cement it into position. The lower portion of the twin-tooth

Fig. 11.—Rods and abutments soldered together at contact points.

Fig. 12.—Twin-teeth cemented to position upon prospective rod sections, forming an all-porcelain bridge.

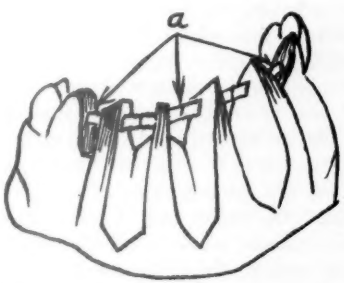

Fig. 13.—Finger-like projections of investment compound holding each individual rod in position before being soldered together at contact points.

(Fig. 2), which bears the force of mastication, though solidly protected by the transverse portion of the rod (Fig. 4, b) and the projecting tongue of the longitudinal portion of the rod (Fig. 4, e), may, under certain circumstances (like biting upon a bone), be broken. Should this occur, the broken portion can easily be replaced by the lower portion of the twin-tooth, which will cost only half what the entire twin-tooth costs. It is then cemented into position without disturbing the rest of the bridge.

5. Economy:

This phase of dentistry can be determined only by comparison. Since this kind of pontic forms an all-porcelain bridge, it is natural and justifiable that we compare it in price with all other all-porcelain bridges of the same catagory. As I have mentioned before, the other two kinds of all-porcelain bridges are:

(a) The one baked on a platinum iridium bar.

(b) The one that is made up of individual jacket crowns cemented on gold or platinum cores.

The twin-tooth pontic to which I am here referring can be commercially produced at approximately $2 a tooth including the rod. The method of constructing such a bridge is very simple, and any practitioner can easily produce it. The amount of gold solder necessary to join the rods of a five-tooth bridge should not exceed more than 4 or 5 grains. The entire cost of the five-tooth bridge should make it readily available to the patient who can afford dental care.

6. Ease and simplicity of construction of a twin-tooth into an all-porcelain bridge:

A plaster impression is made with the abutments in position and a wax bite is taken. A cast is run with a hard investment compound (Fig. 6) and the cast is mounted on an articulator. Twin-tooth pontics are selected as to color and size, adjusted and alined. (Fig. 6, b.) The pontics are adapted to the model, wax being applied to the lingual surfaces. Next, the labial surfaces are embedded in plaster until the plaster crystallizes. (Fig. 6, a.)

The plaster impression is removed from the twin-tooth. (Fig. 7.) The upper section of the twin-tooth is removed, the lower section, including the metallic rods, remaining. The rods and teeth are put in their proper places in the plaster form. (Fig. 8.) The lower sections of the twin-tooth are attached with wax to the plaster form and placed in position on the model. (Fig. 9.) A German-silver wire is placed on the exposed surfaces of the rods (Fig. 9, a.) and the wire allowed to overlap the abutments. The silver wire is attached to the rods and the abutments with sticky wax. (Fig. 10, a.)

The plate wax is broken off the lower section of the twin-tooth, which was attached to the plaster form. (Fig. 8, a.) The plaster form (Fig. 10) is now removed; each lower porcelain section is removed from its rod (Fig. 10), and the rods are invested, the contact points of the rods and also the abutments being omitted. (Fig. 13.) With minute pieces of gold solder, each rod is united at its contact points and also with its prospective abutment. (Fig. 13, a.)

The finished product is now removed from the investment compound and cleaned. (Fig. 11.) The upper and lower portions of the twin-tooth are placed in proper position and the piece is tried in the mouth. (Fig. 12.) After the proper adjustment is made, the bridge is cemented in the mouth.

3460 West Lawrence Avenue.

IMPROVEMENTS AND REFINEMENTS IN LOCAL ANESTHESIA

By J. A. Sweeney,* D.D.S., Cleveland, Ohio

In an era like the past decade, in which there has been rapidly expanding scientific development with many spectacular innovations, some important, though less striking improvements are apt to receive little attention. This applies, it seems to me, to the patient research that has been done in developing more satisfactory solutions for local anesthesia and the refinements in the technic of administering them. Hence, it should be worth while to review these developments and to appraise their value to the dentist and to the public that he serves.

After the discovery of novocain (procaine) and its popularization as an anesthetic, which occurred about 1915, no significant changes or improvements were made in anesthetic solutions for fifteen years. It was generally known that the solutions of novocain used in the early years had a very low hydrogen-ion concentration, or pH; that is, they were strongly acid. Since epinephrine (adrenalin) requires an acid medium to avoid oxidation and deterioration, and since procaine hydrochloride itself is acid, the manufacturers were content to let well enough alone, and were not interested in any change that might interfere with the preservation of the solutions.

These solutions undoubtedly did produce an efficient and long-lasting anesthesia, and those troublesome patients and their dentists who complained about the marked discomfort accompanying the injection and the prolonged pain after anesthesia were reminded that this condition was preferable to no anesthesia at all. In the minds of many patients, the discomforts associated with novocain were so objectionable that they would not permit its use, preferring the immediate pain of the operation to the prolonged discomfort following the injection of the anesthetic. Many insisted on a general anesthetic and thus forced the dental surgeons to rely on gas machines, which, with definite hazards of their own, are, as a rule, definitely less desirable. Now, thanks to improvements in local anesthetics and their administration, many of these apparatus are gathering dust and rust in storage rooms.

The experiments on solutions for use as local anesthetics were begun by Freeman and his associates at Northwestern University in 1931. It was their aim to perfect a solution that would produce rapid anesthesia without pain accompanying, and persisting after, the injection. From the work of these investigators have been evolved the alkaline and the buffered procaine solutions used universally today.

Alkaline procaine solutions have certain definite advantages. They produce anesthesia almost immediately, and cause no pain subsequently. The duration of the anesthesia, however, is shorter than with other solutions, and the solution itself is markedly unstable, since epineph-

Read before the Cleveland Dental Society, May 7, 1941.

*Professor of Anesthesia and Exodontia, Western Reserve University, School of Dentistry.

rine oxidizes rapidly in an alkaline medium. To obtain satisfactory results, a fresh solution must be prepared for each procedure. By some, this might be construed as a disadvantage, but the inconvenience is more than offset by the more satisfactory results and by the saving in cost, as compared with that of prepared solutions.

From the standpoint of the patient, these alkaline solutions are strikingly different in that their use does not cause the cold swollen feeling which accompanies anesthesia with the older acid solutions. To those who associate the presence of this sensation with anesthesia, it often is necessary to explain the difference and to test for sensation before they are convinced that anesthesia is present.

Reputable manufacturers of prepared procaine solutions have been quick to follow the trend of Freeman's research. Since alkaline solutions are too unstable to be commercially practical, they have reduced the acid content of the solutions to a point just short of neutral by adding a buffer salt, usually calcium carbonate. Since the solution remains slightly acid, the epinephrine does not decompose and yet, from a physiologic standpoint, the solutions are more satisfactory because less time is required to convert them to a degree of alkalinity compatible with that of the body fluids. It must be remembered that procaine hydrochloride must be converted to the neutral procaine base before it can become active as an anesthetic.

While use of the buffered solutions does not produce anesthesia so rapidly as when alkaline solutions are employed, because of this short period required for alkalinization they do require considerably less time than the old acid solutions. For some reason, anesthesia with the buffered solutions is longer than that produced by alkaline solutions, even when similar quantities of epinephrine are used. In my clinical experience, the difference in duration of anesthesia and in postinjection discomfort between alkaline solutions and highly buffered solutions is very slight.

One disadvantage has always been associated with the use of procaine solutions, and that is the systemic reaction suffered by some patients after the injection. The patient with such a reaction becomes pale, has a cold clammy sweat and complains of pain in the back just above the region of the kidneys. This condition often terminates in syncope. The picture is typical of shock, and its occurrence is blamed either on the psychic reaction or the action of epinephrine. While it is true that psychic influences are responsible in many instances, and these can be controlled by proper premedication, it is also undoubtedly true that, in some cases, these symptoms are the result of a chemical reaction to the drug. The patient may be susceptible to epinephrine, but the procaine may also be a factor in the reaction. Procaine is a vasodilator, and when the injection is made rapidly, some of the solution may be absorbed systemically.

The use of monocaine, one of the newer local anesthetics, almost never results in any of these troublesome symptoms. It has a synergistic vasoconstricting action with epinephrine so that a greatly reduced quantity of the latter drug is required. Epinephrine with 1 per cent monocaine is administered in a 1 :75,000 solution, while the average concentration with procaine is 1 :20,000 to 1 :50,000. I have used monocaine without any reaction in a great number of cases in which the patients were afflicted with coronary thrombosis or with a marked hypertension. Anesthesia is produced quite rapidly and the intensity is as great as or greater than with procaine.

The medical profession usually suggests the use of cobefrin instead of epinephrine in patients with coronary block or hypertension, on the theory that there

is less danger of increasing blood pressure with its use. The research on vasoconstrictors by Tainter does not substantiate this idea. When injections are made very slowly, the infinitesimal amount of epinephrine is not apt to be absorbed into the circulation because this is prevented by its own vasoconstricting power. It is necessary to recall that only 1:3,200 grain or less to the cubic centimeter of this substance is used in conjunction with a solution for local anesthesia.

It is evident that investigation and research have made available reliable solutions for the production of local anesthesia for all types of patients, and with these at hand, it certainly behooves the profession to give more attention to refinements in their administration. The patient is not convinced that advances have been made if he is apprehensive or if he is hurt by the injection. The patient's criterion of a satisfactory procedure is that the injection be without discomfort, and without physical or mental reaction, and that the operation be painless and have no untoward sequelae. The fulfilment of these requirements may seem impossible at first glance, but there are many procedures which will bring them definitely within the range of possibility.

Every dental patient is naturally apprehensive, but the degree of fear varies with the individual temperament. Many will be calmed and reassured by confidence in the operator and his technical facility, but the highly nervous or neurotic person may require the administration of a hypnotic. Until recently, the dental profession has been handicapped by reluctance to prescribe premedication and also by lack of a drug with a rapid hypnotic action and affording equally rapid absorption without danger of any deleterious effect on the patient. Now education and experience have removed the first barrier and chemical research has obliterated the second.

Pentobarbital sodium, sold under a variety of trade names, fulfils most of the requirements for the dental patient. Unlike the straight barbitals, from which it is derived, its action is apparent within twenty to thirty minutes, and its elimination is but a matter of a few hours. The rapid action of this hypnotic drug is a boon to the busy dentist. The patient is merely advised to appear at the office one half hour before his appointment. The capsule is administered immediately and the patient is instructed to be as quiet as possible, because conversation or reading or any stimulating activity retards or inhibits the action of the drug. For this reason, self-administration of pentobarbital sodium before the patient leaves home is especially contraindicated.

The dose for children ranges from three-fourths to 1 grain and for the average adult is 1½ grains. Highly nervous, neurotic or hyperthyroid patients often require as much as 3 grains to induce complete relaxation. It is much wiser to repeat the average dose, when necessary, than to administer too large a dose at the outset, and thus have a drowsy patient on hand for some time, especially when the office is not prepared for such an eventuality.

When this premedication is to be employed, arrangements should be made for the patient to return home in a cab or to have some one accompany him to drive. If the patient should fall asleep or get drowsy at the wheel and have an accident as the result of administration of a drug by the dentist, the latter would have a moral responsibility, and might possibly be called on to defend a legal action.

The appearance of the patient who receives pentobarbital sodium will vary greatly. Those with low blood pressure, the weak and debilitated and the aged will require only the minimum dose and even then may show marked signs of drowsiness. Occasionally, they have to be

assisted to the dental chair. On the other hand, patients with highly nervous temperaments may show no sign of hypnosis even when 3 grains has been administered. The great average group show no outward symptoms, but will be greatly relaxed mentally and display a slight lassitude. The degree and action of any hypnotic drug is directly dependent on the basal metabolic rate of the patient. Since the evaluation of this rate involves a complicated test, the dosage must necessarily be approximate and must be gaged by personal experience.

With our realization of the tremendous advances made by the dental profession in the avoidance and prevention of pain, it is often impossible to comprehend the mental attitude of some of our patients. Their anticipation of the pain which may result from a dental procedure magnifies its importance a hundred fold, while to the dental surgeon it is merely a matter of routine. Of course, the situation seems somewhat different when the dentist himself is the patient. It may be this utter objectivity in connection with our own work that makes us overlook or neglect the many niceties in technic that would minimize or eliminate much of the attendant discomfort to the patient. The confidence of the patient can be lost immediately when an injection is made with a dull or barbed needle, and every effort of the dentist may from then on be regarded with suspicion and increased tension.

Needles should be sharpened frequently and care should be taken that any barbs be removed from the opposite side of the bevel. The presence of these barbs results in a drag on the tissues much like the action of a saw. After the honing or sharpening is completed, the needle point should be inspected with a magnifying glass. It is amazing how many defects may thus be discovered, even in new needles. It is our experience that it is not the size of the needle, but the dullness of the point that is responsible for much of the initial pain of insertion. For deep nerve block, a 23 or 25 gage needle should be used; but for the infiltration injections, the 25 gage or even the very fine 27 gage is preferred.

The use of a sharp, long-bevel needle and the application of an effective topical anesthetic to the point of injection will prevent the initial pain. Experiments have proved that 10 per cent butyn or benzocaine are the best anesthetics for topical use. Phenol is recommended by some, but there is always the possibility of a painful surface slough following its use. The mucous membrane should first be dried, so that the surface tension of the saliva shall not prevent absorption, and the topical anesthetic must be held in contact with the tissues for at least a minute or two before its action is discernible.

The foregoing procedures designed to prevent pain can go for nought through failure to warm the syringe and the solution to body temperature. The contact of cold metal and solution may shock the patient sufficiently to affect his composure. A like condition may also be noted when the solution, instead of being injected slowly, drop by drop, is suddenly forced into the tissues. This can be avoided by freeing the plunger and expelling a few drops of solution before the needle is inserted.

The mucous membrane should be drawn taut and the needle inserted with a gliding motion, not with a pronounced push or jab. To accomplish this, the syringe should be held loosely in pen fashion, much as an artist would hold his brush. This method affords a delicacy of feeling which cannot be achieved when the syringe is held tightly in the palm of the hand.

It is impossible to lay too much stress on the importance of slow injection. In subperiosteal infiltration, rapid injection balloons the periosteum from the surface

of the bone, with resultant pain, and fast nerve block injections allow the solution to be absorbed systemically before the vasoconstricting solution can act effectually. Contrary to the opinion of some operators, slow injection is not a waste of time, for the first portion of solution injected is already producing anesthesia when the needle is withdrawn.

Self-assurance and delicacy of technic on the part of the operator add greatly to the patient's confidence in him. This can be supplemented by the use of a little psychology in proving the presence of anesthesia to his complete satisfaction before any operative procedure is attempted.

The use of the positive rather than negative approach has the best results. In the former, the difference between pressure and pain is thoroughly demonstrated and the patient is assured that while he will feel the sensation of pressure, pain will be entirely absent. Tests are then made on the anesthetized area to prove the point. In the latter, or negative method, the patient is asked whether pain is felt. This apparent uncertainty on the part of the dentist fills the patient with doubt and apprehension. His confidence is lost and he is constantly waiting, and expecting, to be hurt. The resultant tension is transmitted to the operator and the result of the dental procedure may be unsatisfactory.

A positive manner and positive suggestions cannot be conveyed successfully by a dentist who has little or no confidence in his own technic. Until he has mastered every detail of the procedure and has a facility based on a thorough knowledge of anatomy and neurology, the doubt lurking in his own mind will be apparent in his actions.

The science of local anesthesia has reached heights which would have seemed impossible ten years ago, and improvements in this field are largely responsible for the present high standards of dentistry. In realizing its effectiveness, however, its limitations must be carefully evaluated. Disregard of the limitations and contraindications result in troublesome complications and may unjustly discredit much that has been accomplished.

Although every one who has taught, or written about, local anesthesia has repeated again and again that injections should not be made into swollen, inflamed and infected areas, especially when the infection is of the diffuse type, it still is no uncommon thing for this to be done. In these cases, the unpleasant train of pain, swelling, cellulitis, etc., often is blamed on the anesthetic rather than on the judgment of the operator, where it belongs.

With the perfection of improvements and refinements in local anesthesia, it should be possible to perform painless operations in all cases wherein this type of anesthesia is indicated, but it must always be remembered that anesthesia is an inanimate agent and that it requires the guiding hand of an intelligent master.

BIBLIOGRAPHY

BENEDICT, H. L.; CLARK, S. W., and FREEMAN, C. W.: Studies in Local Anesthesia. J.A.D.A., 19:2087-2105, December 1932.

TAINTER, M. L.; THRONDSON, A. H., and MOOSE, S. M.: Comparison of Procaine Hydrochloride and Procaine Borate (Borocaine) as Local Anesthetics. J.A.D.A., 24: 376-386, March 1937.

CLARK, S. W.: Research in Local Anesthesia and Its Clinical Application. J.A.D.A., 23:251-254, February 1936.

TAINTER, M. L.: Causes in Systemic Reactions in Local Anesthesia. J.A.D.A., 20: 152-155, January 1933.

TAINTER, M. L., and THRONDSON, A. H.: Choice of Vasoconstrictor in Local Anesthetic Solutions. *Modern Dent.*, 7:7-9, October 1940.

710 Rose Building.

COMPOUND AND COMMINUTED FRACTURE OF THE RAMUS

REPORT OF CASE

By Edward J. Durling, D.M.D., Boston, Mass.

History.—C. W., a boy aged 8 years, was in an automobile accident April 8, 1939, the handle of the car catching the corner of the mouth. He was removed to a house where cold cloths were applied until the ambulance arrived.

Examination.—The patient was conscious.

Fig. 1.—Appearance of the patient upon arrival in operating room.

There was a deep laceration extending from the angle of the mouth downward to below the jaw and extending back to the ear. It involved about 1 inch of the oral mucous membrane, which was severed for about 1 inch from the corner of the lips. The wound gaped widely and pieces of mandible could be seen in the wound. The laceration was deep, involving the facial muscles and extending downward to the mucosa. The patient could move the jaw and area about the wound normally.

Preliminary Diagnosis.—Laceration of the face, with compound and comminuted fracture of the ramus of the mandible.

Operations.—April 8, a soft tissue repair was made by Herbert G. Dunphy, M.D. Under gas-oxygen and ether, the wound was cleansed with green soap and water, and irrigated with 1,500 cc. of a physiologic solution of sodium chloride. The wound was then debrided and the skin prepared with an antiseptic solution. The mucous membrane of the mouth was sutured and then the obicularis oris muscle. The deep muscles of the cheek were reunited. The upper part of the parotid gland lay exposed in the wound. A piece of bone at the angle of the jaw which was entirely detached was removed. The jaw bone was alined and the periosteum sutured over the fragments. Superficial fascia was sutured in place with No. O. plain suture and the skin closed with multiple fine silk sutures closely placed. One piece of skin resembling a full thickness graft about the size of a nickel was replaced with the hope that it would be reattached and a skin graft would be unnecessary.

Leroy M. S. Miner, on emergency call, fixed the fracture temporarily by intermaxillary wires on the deciduous central incisors, bringing the teeth into occlusion, but with some question as to the position of the posterior fragment. He advised further x-ray treatment, with probably the use of other agents after the swelling and acute condition subsided, for complete reduction.

While the patient was being operated on, angle for about three-quarters inch. A flat periosteal elevator was inserted behind the angle and a hole about one-half inch from the border was made with a large round bur. A double strand of Angle's 0.020 inch ligature wire was inserted and drawn out straight posteriorly. This was attached to a hook on the headgear, by means of a common elastic band. (Darcissac's method [Ivy and Curtis: Fractures of the Jaws].)

Fig. 2.—Intermaxillary wiring; showing posterior fragment in poor position. (April 28.)

600 cc. of physiologic solution of sodium chloride was given by clysis. In the ward, 500 cc. of 5 per cent dextrose in a physiologic solution was given intravenously.

The patient left the table in fair condition in spite of two and one-half hours' anesthesia. Owing to the condition of the soft tissue, it was decided to wait some time before attempting further reduction.

April 27, under gas-oxygen-ether anesthesia, intermaxillary wires were placed on the right and left deciduous molars and anterior central incisors. The jaws were in centric occlusion. An attempt was made to push the ramus into position, with doubtful success. Further x-ray treatment with reduction through the use of headgear and gradual traction later was advised.

May 2, under gas-oxygen-ether anesthesia, an external incision was made through the skin and fascia just under the right angle of the jaw. The masseter muscle and periosteum were peeled away from the lower posterior border of the ramus to expose the

Fig. 3.—Patient with common type headgear (designed by Dr. Miner and used with various attachments for most cases requiring headgear.)

During anesthesia, it was found through manipulation that the joint was movable, this indicating no dislocation. There was movement of the fragments and some reduction was accomplished at this time. The inter-

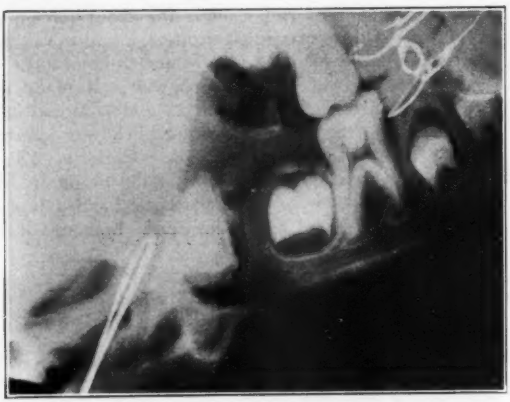

Fig. 4.—Application of traction, May 13.

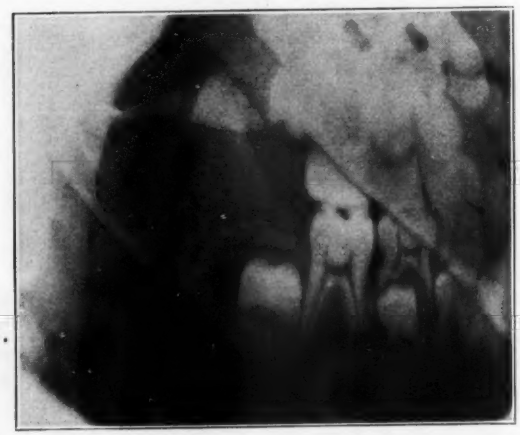

Fig. 5.—X-ray appearance October 31.

maxillary wires which had been disconnected during this procedure were connected, with the teeth brought to normal occlusion.

Outcome.—The boy remained fairly comfortable and the wound was in good condition except for slight drainage at times.

June 5, under gas-oxygen anesthesia, the remaining intra-oral and extra-oral wires were removed. The fragments were in good position and, after a few days, the jaw opened normally with no deviation from the normal central line.

Fig. 6.—Resultant scar, October 31.

COMMENT

This is the third case that I have re-

cently treated in this manner, the present case being the most successful one. One of an edentulous type was reasonably successful. In another, the bones were held in apposition, but the fragments were not in quite so good a position. Probably the time of operation and the age of the patient were the determining factors.

363 Marlborough Street.

VINCENT'S INFECTION

By BERNARD S. CHAIKIN,* D.M.D., Boston, Mass

VINCENT'S infection, a disease originating from the fusospirochetal organisms, is much more prevalent than is commonly recognized. The disease may be transmitted from one individual to another; but a completely healthy mouth, which is found to have no trace of gingivitis upon thorough examination, seldom becomes infected. This would seem to indicate that, in healthy areas, where the resistance to bacterial invasion has not been lowered, the mucous membranes have sufficient resistance to prevent the penetration and growth of bacteria—especially in the vulnerable, protected areas such as the interdental spaces and the retromolar flaps. As a matter of fact, it is not uncommon to obtain a positive smear from the same sheltered areas, and, at the same time, find no signs of Vincent's infection upon inspection of the mouth.

The disease, which, as has been said, may be transmitted or self-infective, is generally superimposed on conditions varying from a slight marginal gingivitis to an advanced periodontoclasia, according to the tissue resistance, which, as it becomes lowered, allows a more fluent growth of the organisms. In infrequent cases, a patient may be unaware of, and may experience no discomfort from, the disease, and yet its presence may be disclosed by a thorough examination.

*Periodontist, Forest Hills Hospital.

The many forms of Vincent's infection may be generally cataloged in two distinct groups, the acute and the subacute. The infection always has its beginning as the acute form, either in a mild or in a severe degree; but lack of treatment, or perhaps cessation of treatment before the tissues have returned to normal, allows it to progress to the subacute type. Patients suffering from subacute, or chronic, Vincent's infection too often recall painful mouth lesions of many years before, when dental services were discontinued too early to effect a complete cure, or perhaps advertised mouthwashes were used as an ineffectual home remedy.

Acute Vincent's infection, more correctly termed acute ulcerative stomatitis, is generally of a painful nature. Its symptoms are easily recognized, and therefore are seldom erroneously diagnosed. It perhaps most often comes to the attention of the general practitioner either just before or just at the time its symptoms, which develop suddenly, have reached their full development. In mildly acute cases, there are slight ulcerative spots in the retromolar flaps or in isolated areas in the interdental spaces. Such areas are covered with a layer of necrosed tissue, which may be gently wiped away to reveal a painful, highly inflamed surface, bleeding profusely at a slight touch. Symptoms are rarely evident throughout the entire mouth at

one time, except that the patient often complains of a disagreeable metallic taste, and there is always a distinctive offensive odor to the breath, which, once encountered, will never be mistakenly diagnosed.

If the areas of infection are the septal papillae, they may entirely disappear.

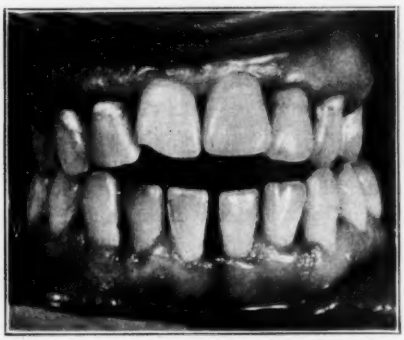

Fig. 1 (Case 1).—Gingivae showing marked interdental pockets between upper right and left cuspids and lateral incisors, and lateral and central incisors; lower right and left cuspids and lateral incisors, and lower central incisors.

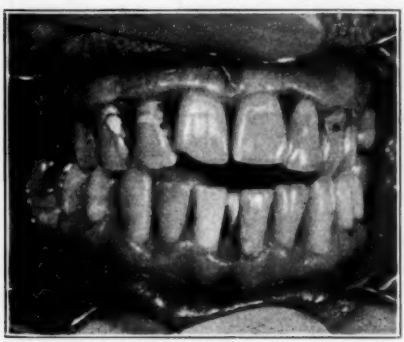

Fig. 2 (Case 1).—Appearance after treatment. The interdental pockets have been closed by proliferation of epithelium, forming a convexity labiolingually instead of a deep concavity.

Should the case be allowed to progress untreated, these areas will become increasingly painful, and the diminished use of the toothbrush will allow the disease to spread from the interdental areas to the labial, buccal and marginal gingivae, and thus gradually develop into the severe active, acute stages, and then possibly subside to the chronic, or subacute, type.

In the more severe type of acute infection, the symptoms develop gradually rather than suddenly, and may be first noticed by the patient as swellings of either the submaxillary or the sublingual

Fig. 3.—Elongated papillae that can be lifted away from teeth, pockets thus being exposed, bases of which are covered with necrotic layer overlying raw surface that bleeds and is painful to touch.

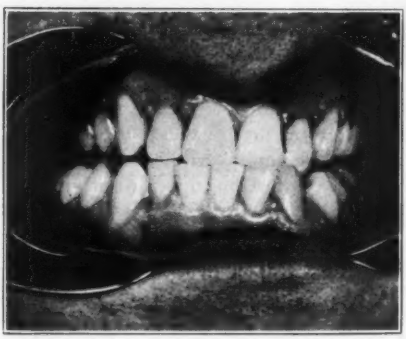

Fig. 4 (Case 2).—Bulbous interdental papillae. Here, as in Figure 3, when the papillae are lifted, pockets from 3 to 5 mm. in depth are exposed. There is a shortened papilla between the lower right cuspid and lateral incisor, where there is a pocket. This is a common site for disease.

glands (or perhaps both), small ulcerative spots on the cheeks, palate or gingivae and a temperature ranging from 101 to 103° F. or perhaps higher. Within a day or two, the ulcerative spots blend into each other to produce the peak symptoms, which are unmistakably ulcerative stomatitis. Patients thus afflicted are generally quite ill. They may be dehydrated and have tendencies to constipation, malaise, vomiting and a general lack of appetite.

The indication here would be to confine the patient to bed until the temperature had subsided to normal and to instigate corrective treatment immediately.

The second, or subacute, type of Vincent's infection presents varied symptoms which are recognized only by the experienced diagnostician, and are often completely ignored by the general practioner. This type may, in its turn, be separated into two groups, the destructive and the proliferative.

The destructive group assumes many forms and presents varying symptoms. Hirschfeld, in his article "Vincent's Infection of the Mouth: Clinical Incidents in Its Diagnosis and Treatment," lists seventeen different clinical and roentgenographic signs, but I shall confine myself here only to those most frequently seen in everyday practice.

The most common sites of infection, as has already been cited, are the protected interdental and retromolar spaces. Here, generally, is found the grayish, necrotic tissue, the removal of which will disclose the underlying raw, bleeding, tender surface. The papillae may become necrotic and slough away, only the interdental concavities (seldom found labially, buccally or lingually) remaining, with labial and lingual flaps, and the mesial and distal walls of which are the denuded roots of the proximal teeth. (Fig. 1.) The labial and lingual flaps may become bulbous, elongated or edematous, so that they can be lifted away from the tooth with a probe. (Figs. 3 and 4.) The tips of the papillae may be of uneven height (Fig. 8); nor is it unusual to detect a growth of one papilla incisally; while others in the same mouth appear blunt and shortened to varying degrees (Fig. 7), each one seeming to form a sort of truncated cone. (Figs. 5 and 7.) An inflamed area, extending

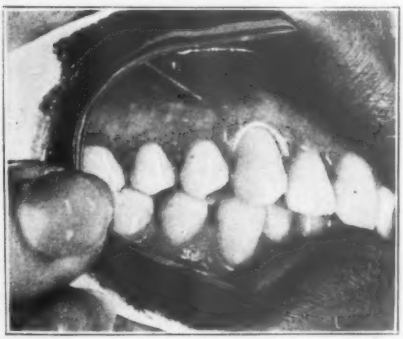

Fig. 5 (Case 2).—Right lateral view. The papillae between the upper right cuspid and the first bicuspid, the first and second bicuspids and the second bicuspid and the first molar all have their tips rounded, which gives them a truncated cone effect. This condition, coupled with pockets in these areas having tender bleeding necrotic bases, is a definite symptom of subacute Vincent's infection.

Fig. 6 (Case 2).—Appearance after treatment. The pockets have been eliminated by the shrinking of the hypertrophic papillae.

rootward, will perhaps be in evidence. (Fig. 7.) Necrosed papillae have even sloughed away, to disappear entirely, leaving merely a straight line as the outline of the marginal gingivae. (Fig. 1.)

Each one of these symptoms is important of itself, and although they may be seen singly or as a combination of several, no one symptom should ever be allowed to go untreated, as its destructive processes are progressive, lacking interference.

In the proliferative form or, as it is sometimes known, the hypertrophic form of subacute Vincent's infection, the septal gingivae may be enlarged to such an extent that two engorged papillae may meet to form a cleft on the labial or lingual surface of the tooth that they surround. (Fig. 9.) A hypertrophic septum may even force a surrounded tooth out of position, although, in some instances, the shrinking action (after treatment) of the healing gingivae may draw the tooth back into position again. Hypertrophied areas are generally spongy, with a tendency to bleed easily at a slight touch.

The diagnostician should be extremely careful in dealing with these cases to differentiate between these and other conditions having similar symptoms, which may perhaps be caused by blood dyscrasia, menstruation, pregnancy, etc.

The following cases of subacute Vincent's infection present most of the symptoms of this disease.

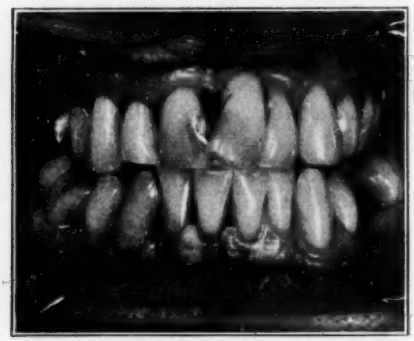

Fig. 7.—Interdental pockets with necrotic, tender bases. (Compare Figs. 4-6.) The papilla between the lower left central and lateral incisors is elongated and can be lifted away from the teeth for its whole length. The tips of the papillae between the lower right lateral and central incisors and between the lower central incisors are rounded, having the appearance of truncated cones. The dark longitudinal areas between the lower central incisors and the lower right cuspid and lateral incisor are actually areas of inflammation, denoting pocket formation. Here again, the papilla between the lower right cuspid and lateral incisor is shortened and punched out, with pocket formation. All of these conditions are pathognomonic of subacute Vincent's infection.

Fig. 8.—Tips of papillae between lower incisors of uneven height. They can be lifted away from the teeth, when necrotic tissue overlying raw, bleeding and tender bases is exposed. These are definite symptoms of subacute Vincent's infection. (The black line around the lower left central and lateral incisors indicates hemorrhage due to drying the tissues for the photograph.)

REPORT OF CASES

CASE 1 (Fig. 1).—L. S., aged 30, a traveling salesman, presented a subacute Vincent's infection of the destructive type. Examination of his mouth revealed interdental pockets 8 to 10 mm. in depth. No labial, buccal or lingual pockets were in evidence. The bases of these pockets were sensitive to the probe and were covered with necrotic tissue, which, when wiped away, left the typical raw, bleeding surface. The patient was a heavy smoker.

His breath was offensive, and he complained of a constant disagreeable metallic taste. He related also that, approximately four or five years previously, he had developed an acute case of Vincent's infection, which had been treated superficially. Since that time, he noticed that the symptoms were growing progressively more severe. He sought advice and treatment from several practitioners, only to be told that the only remedy was to have all of his teeth extracted. This, naturally, he hesitated to have done, and of course I reassured him as to its inadvisability. Corrective treatment was instituted immediately, with the gratifying results that are evident in the accompanying photographs. (Fig. 2.)

CASE 2 (Figs. 4 and 5).—R. S., a student, presented a destructive subacute Vincent's infection. The tips of the papillae between the upper incisors were bulbous and could be lifted away from the teeth to disclose pockets from 3 to 5 mm. in depth. The bases of these pockets were very sensitive to the probe. The tips of the papillae between the upper right cuspid and first premolar, and the first and second premolars between the upper right first molar and second premolar, and between the lower right lateral incisor and cuspid were all rounded, to suggest a sort of truncated cone. The patient had been troubled with consistent bleeding of the gums, and although various dentists had treated him, the ultimate diagnosis of each was that nothing could be done

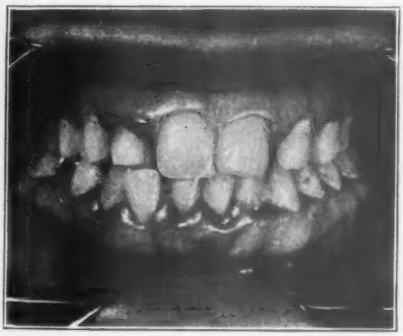

Fig. 9 (Case 3).—Proliferative type of subacute Vincent's infection. The interdental papillae in the lower incisor area and the upper right lateral area are hypertrophic and filled with stagnant venous blood, and are therefore markedly cyanotic. These papillae bleed freely and may be lifted away from the teeth and the pockets and necrotic tissue overlying tender bleeding bases exposed. The lower right lateral incisor was forced out of position by the pressure of the engorged papillae mesially to it. The papilla between the lower left lateral incisor and cuspid was electrically cauterized by a physician a week previous to the time that the photograph was taken. These areas are often diseased because of the difficulty many people have in reaching them with a toothbrush.

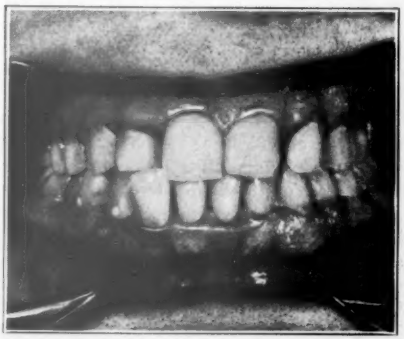

Fig. 10 (Case 3).—Appearance after treatment. The papillae have shrunk to normal size and in doing so have pulled the lower right lateral incisor almost into line.

Fig. 11 (Case 4).—Active phase of proliferative type of subacute Vincent's infection. The papillae can be lifted away from the teeth and a pocket filled with necrotic tissue exposed. When this tissue is wiped away, a raw, bleeding surface remains, very tender to the touch of a dull probe.

for him. Anti-Vincent's treatment was instituted, followed by thorough subgingival curettage. The results are evident in the final photographs. (Fig. 6.)

CASE 3 (Fig. 9).—G. L., a woman, aged 23, presented a hypertrophic gingivitis of four years' duration. The papillae were loose enough to be pulled away from the teeth, with exposure of pockets, the bases of which were covered with necrotic tissue, were painful and bled freely when touched with the probe. The papilla between the lower right lateral incisor and central incisor was so edematous that it forced the lateral incisor out of position. The papilla between the lower left lateral incisor and cuspid had been cauterized electrically by a physician shortly before this photograph was made. The lower incisal and upper right lateral areas of the mouth were affected most severely. Right-handed persons quite frequently present this same infected area, since it is difficult to reach with the toothbrush and therefore suffers from unwitting neglect. Oftentimes, as in this case, the firm lower lip muscles will reject the toothbrush in its attempted massage of the lower anterior region, with resultant infection of the gingivae.

It is interesting to note that corrective treatment of the gingivae surrounding the lower right lateral incisor caused the gingivae to shrink to normal, drawing the lateral incisor, which the edematous gums had forced out of position, back almost into line. (Fig. 10.)

CASE 4 (Fig. 11).—E. M., a girl, aged 20, presented an active subacute Vincent's infection of the proliferative type. The history is typical of the cases which are alternately active and quiescent for a period of months or perhaps years. In this case, the infection was of two years' duration, at the end of which time the patient sought treatment only because bleeding was becoming more profuse. Examination revealed that the papillae throughout the entire mouth were inflamed and hypertrophic and could be lifted away from the teeth to reveal pockets filled with necrotic tissue and débris. The tips of all the papillae were rounded to the truncated cone shape, while those in the lower anterior region were of varying heights. The breath was very fetid.

None of these cases described have presented unusual or unrecognizable symptoms. That there are a rapidly increasing number of cases of Vincent's infection may be attributed to the fact that many men are unfamiliar with its signs. Nor are they cognizant of the irreparable destruction that it may cause. The ultimate solution of this problem is to familiarize the general practitioner with the many forms of Vincent's infection, its causes and their correction; for just as soon as a correct diagnosis is obtained and a course of treatment decided upon, the only essential requirement for a complete cure is painstaking thoroughness and stubborn persistence.

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SUBACUTE BACTERIAL ENDOCARDITIS AND DENTAL EXTRACTION

By A. Henry Clagett, Jr., M.D., M.Sc. (Med.), Wilmington, Del., and Edwin H. Smith, Jr., D.D.S., Washington, D. C.

SUBACUTE bacterial endocarditis has been, from the therapeutic standpoint, a major enigma in the practice of medicine. As in cancer, there has been much work done in regard to the pathogenesis and therapy of the condition. The pathogenesis, unlike that of cancer, is well understood, but the therapy, so far, has been of no avail. Nearly every chemotherapeutic agent has had, at one time or another, a clinical trial in the treatment of this disease. Soon after its discovery, arsphenamine was tried in the treatment of this disease, but it was found to be of no definite benefit. Within the past few years, it has again been tried, this time in combination with sulfanilamide, but again the results have not been remarkable.

The latest and most logical regimen suggested for the treatment of this fatal disease has been the combination of sulfapyridine with heparin.[1] Sulfapyridine is known to sterilize the blood stream in cases of *Streptococcus viridans* bacteremia. However, this alone is not sufficient because, in bacterial endocarditis, thrombotic lesions cover the valvular lesions and protect some of the causative organisms from the action of the chemotherapeutic agent and leukocytes present in the blood stream.[2] Therefore, heparin, an anticoagulant, was recommended, together with sulfapyridine, to prevent formation of thrombi and perhaps to do way with those already present and therefore allow the drug and leukocytes to reach all of the pathogenic organisms. This, as mentioned previously, is the best treatment to date, but, unfortunately, unforeseen complications, such as cerebral hemorrhage due to an excessively prolonged bleeding time, made the treatment almost as hazardous as the disease itself. It may be said, therefore, that, for all practical purposes, the disease is 100 per cent fatal.

The theory expressed in this paper regards the prevention of the disease in some cases and is based upon the following generally recognized facts.

Subacute bacterial endocarditis rarely, if ever, affects a normal valve. It is, for all practical purposes, always superimposed on a crippled condition of the valve, usually the result of rheumatic disease. However, this condition is also found to be the major complicating factor in congenital cardiac defects.

The pathogenic organism in this type of endocarditis is usually *Streptococcus viridans,* commonly found in oral infections, especially around the apices of infected teeth. Therefore, the first thought is to rid the mouth of all infected teeth. However, it has been shown that a transient bacteremia following dental extractions is not uncommon,[3, 4, 5] occurring mostly in the case of septic mouths, but also occurring to an appreciable degree in clean mouths, as shown in the accompanying table.

By far the predominant organism causing this bacteremia is *Streptococcus viridans.*[4] It has also been shown that merely the manipulation of a tooth, without extraction, is sufficient to cause

a bacteremia,[6] usually of ten minutes' duration only. While such a transient condition could be well tolerated by a normal person, it cannot in the presence of a valvular heart lesion. There are a number of cases reported in which subacute bacterial endocarditis followed dental extraction.[3-5, 7-13] To further complicate the problem, foci of infection, as is already known, if left undisturbed, will sooner or later cause a bacteremia.[3] Therefore, it seems that our hands are tied and that a bacteremia will result if the focus is removed, if it is manipulated or if it is left alone. However, the fact that a definite focus of infection should be removed is unquestioned.

RELATIONSHIP OF BACTEREMIA TO DENTAL PROCEDURES

Author	Operation	Mouth Condition	No. Cases	Bacteremia
Palmer and Kempf	Extraction	Normal	82	14*
Richards	Manipulation; gum massage	Septic	17	3
O'Kell and Elliott	Extraction	Septic	138	84
O'Kell and Elliott	No operative procedures	Septic	110	12

*Of the fourteen blood cultures in this series thirteen were due to the presence of *Streptococcus viridans*.

Sulfapyridine has been shown to remove *Streptococcus viridans* from the blood stream. In cases of established subacute bacterial endocarditis, this elimination is only temporary.

Hageman,[14] in 1940, suggested the prophylactic use of sulfanilamide in cases of dental extraction in persons with valvular heart disease. This seems to be the most logical approach to this problem, at least when, in subacute bacterial endocarditis, bacteremia is due to dental foci. Hageman mentioned this use of sulfanilamide as a possibility, but laid down no definite plan of attack.

We have formulated the following plan as the one most likely to have the desired effect in the majority of cases.

In all extraction cases, the dental surgeon should attempt to ascertain whether valvular heart disease is present. This can be accomplished by (a) medical consultation, (b) reference to previous medical records or (c) direct questioning of the patient. The first method is the most desirable, but is impractical in that it is impossible to have a consultation in every extraction case. The second method is excellent, but its use is not always possible. The third method is the one which must be used in the majority of cases.

With a few well-selected questions, the surgeon can obtain a cardiac history. An intelligent person can often save much useless questioning by immediately supplying the information as to whether heart disease is present, such information having been obtained from a physician. However, if the patient cannot supply such information, he should be questioned as to the presence of cyanosis, edema, dyspnea and precordial distress. It is also well to ask whether he was a "blue baby" and whether there is evidence of any of the rheumatic group of infections. The patient should be specifically interrogated concerning rheumatic fever, scarlet fever, chorea, epistaxis and growing pains. A positive answer to any of these questions is sufficient to warrant a medical survey prior to dental extraction.

With the demonstration of the presence of valvular heart disease, the patient becomes the dual responsibility of the dental surgeon and the internist.

Ideally, the patient should be hospitalized prior to the extraction. It cannot be stated with certainty just when the patient will be ready to have the extraction, as this depends on the concentration of the sulfapyridine in the blood stream. The administration of the drug is the sole responsibility of the internist.

It is also his duty to state the optimum time for extraction. To attain the desired blood level of 6 mg. per hundred cubic centimeters, it is necessary to give about 6 gm. (average) of sulfapyridine daily. However, this dosage is variable and the two criteria of proper dosage are the reaction of the patient and the blood level of the drug. It is for this reason, that is, to insure close observation and control of the patient, that hospitalization is recommended.

Usually, the blood sulfapyridine concentration reaches the desired level on the second day of administration. When this is attained, the extraction may be effected, a proper surgical technic (cleanliness, minimum of trauma) being taken for granted.

The selection of the anesthetic agent is of some importance. Most observers agree that the only anesthetic agents contraindicated in the presence of sulfapyridine are the intravenous barbiturates.[15-17] This combination is supposed to cause sulfhemoglobinemia about five days after the combination is administered. This point is disputed. However, with many other anesthetic agents from which to choose, one can forget the intravenous barbitals.

The actual administration of the sulfapyridine is the responsibility of the physician. However, the length of time that the blood level of 6 mg. per hundred cubic centimeters or more must be maintained is to be determined by the dental surgeon. In the experimental work previously quoted, the transient bacteremia following extraction lasted only ten minutes. It is therefore true that, in cases which react normally to extraction, continuation of this dosage for one or two days after the operation will probably be ample. However, in cases of dry socket, the source of infection may continue to be active for some time. Therefore, no definite rule can be laid down, but each case must be treated according to its individual reactions.

SUMMARY

1. Subacute bacterial endocarditis is caused by *Streptococcus viridans,* which does not attack a healthy cardiac valve.

2. *Streptococcus viridans* is, in a great number of cases, the causative agent in a transient bacteremia following dental extraction. This may happen even in a healthy mouth. It has also been demonstrated in the blood stream prior to extraction and also after manipulation of a tooth without extraction.

3. Sulfapyridine has been shown to be capable of sterilizing the blood stream when the bacteremia is due to *Streptococcus viridans.*

4. It is suggested that in cases of known valvular heart disease sulfapyridine be given prior to dental extraction.

CONCLUSION

It is hoped that by use of the method outlined or a similar procedure, the bacterial endocarditis following dental extraction will be prevented. It is realized that, with the relatively low incidence of subacute bacterial endocarditis, it will take many years to determine the value of this procedure. The method, in proper hands, is harmless, and, because it seems efficacious in the prevention of a fatal disease, should be given a widespread trial. Hope for the success of this method lies in close cooperation between the dental surgeon and the physician.

BIBLIOGRAPHY

1. KELSON, S. R.: New Method of Treatment of Subacute Bacterial Endocarditis Using Sulfapyridine and Heparin: Preliminary Report. *J.A.M.A.,* 113:1700, November 4, 1939.

2. FRIEDMAN, M.; HAMBURGER, W. W., and KATZ, L. N.: Use of Heparin in Subacute Bacterial Endocarditis: Preliminary Report. *J.A.M.A.,* 113:1702, November 4, 1939.

3. OKELL, C. C., and ELLIOTT, S. D.: Bacteremia and Oral Sepsis with Special Reference to Etiology of Subacute Bacterial Endocarditis. *Lancet,* 2:869, October 19, 1935.

4. PALMER, H. D., and KEMPF, M.: Streptococcus Viridans Bacteremia Following Extraction of Teeth; Case of Multiple Mycotic Aneurysms in Pulmonary Arteries: Report of

Cases and Necropsies. *J.A.M.A.*, 113:1778, November 11, 1939.

5. HOPKINS, J. A.: Streptococcus Viridans Bacteremia Following Extraction of Teeth. J.A.D.A., 26:2002, December 1939.

6. RICHARDS, J. H.: Bacteremia Following Irritation of Foci of Infection. *J.A.M.A.*, 99: 1496, October 29, 1932.

7. CALVY, P. J.: Dental Surgery and Organic Heart Disease. *J.A.M.A.*, 74:1221, May 1, 1920.

8. BERNSTEIN, M.: Subacute Bacterial Endocarditis Following Extraction of Teeth: Report of Case. *Ann. Int. Med.*, 5:1138, March 1932.

9. BROWN, H. H.: Tooth Extraction and Chronic Infective Endocarditis. *Brit. M. J.*, 1: 796, April 30, 1932.

10. ABRAHAMSON, L.: Subacute Bacterial Endocarditis Following Removal of Septic Foci. *Brit. M. J.*, 2:8, July 4, 1931.

11. VANDERHOOF, D., and DAVIS, D.: Subacute Bacterial Endocarditis Following Extraction of Teeth, with Report of Two Cases. *Virginia M. Month.*, 60:151, June 1933.

12. VON PHUL, P. V.: Subacute Bacterial Endocarditis; Three Cases Following Extraction of Teeth. *Northwest Med.*, 32:188, May 1933.

13. FELDMAN, L., and TRACE, J. M.: Subacute Bacterial Endocarditis Following Removal of Teeth or Tonsils. *Ann. Int. Med.*, 11:2124, June 1938.

14. HAGEMAN, P.: Use of Sulfanilamide. J.A.D.A., 27:909, June 1940.

15. ADRIANI, J.: Effects of Anesthetic Drugs upon Rats Treated with Sulfanilamide. *J. Lab. & Clin. Med.*, 24:1066, July 1939.

16. KING, W. L. M.: Evaluation of Combined Effects of Sulphapyridine and Barbiturates in Treatment of Pneumonia. *Proc. Staff Meet., Mayo Clin.*, 15:21, January 1940.

17. LUNDY, J. S., et al.: Annual Report for 1938 of Section on Anesthesia, Including Data on Blood Transfusion. *Proc. Staff Meet., Mayo Clin.*, 14:273, May 3, 1939.

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THE COMPARATIVE VALUE OF VARIOUS MEDICAMENTS IN CAVITY STERILIZATION

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THE dental profession has not given much attention to the subject of dentin sterilization. Heretofore, we have based the value of medicaments on testtube standards such as the phenol coefficient. A review of the literature has failed to reveal much research on dentin sterilizing agents. Miller[1] (1891) outlined four methods for testing the action of antiseptics on carious dentin. None of these methods was based on clinical work. Miller concluded that water-soluble solutions were most efficient as germicides in cavity sterilization. In 1935, Barker[2] tested the action of various antiseptics on slabs of dentin infected *in vitro* with organisms from carious cavities. He found that Howe's ammoniacal silver nitrate was the most effective of all the antiseptics tested.

The present study is an attempt to find out (1) which drugs are valueless in cavity sterilization and therefore can be eliminated from the armamentarium for this purpose, and (2) which drugs actually sterilize dentin. It is realized that, from the work done here, a final conclusion cannot be reached regarding the efficiency of any drug until other important points, such as the effect on the pulp, have been determined. However,

Work done at the Children's Dental Clinic of the Philadelphia Mouth Hygiene Association; facilities provided for the investigation by the Philadelphia Mouth Hygiene Association.

from the standpoint of sterilization alone, certain conclusions can be drawn. In this study, drugs are evaluated on the basis of their sterilizing action in the cavity. In other words, this is a bacteriologic check-up, *in situ*, on the efficiency of medicaments used in cavity sterilization.

PROCEDURE

The work was confined to occlusal cavities, since these could be sealed more easily and with more assurance that the temporary cement would not break under mastication. All teeth under observation were placed under a rubber dam. The teeth and the adjacent dam were painted with tincture of iodine followed by alcohol and chloroform. Thereafter, every instrument or bur to be introduced into a cavity was dipped in a molten metal sterilizer and allowed to remain for twenty seconds. Appleton[3] has shown that this procedure will sterilize an instrument. The cavity was then prepared and all carious material removed with a sharp sterile excavator. The cavity was prepared exactly as it would be for a metallic restoration. As soon as it was felt that the cavity was ready for filling, but prior to the introduction of any medicaments, shavings from the dentin at the floor of the cavity were immediately inoculated into a tube of Rosenow's brain broth, which is excellent for the growth of streptococci and lactobacilli and has the additional advantage of growing aerobes and anaerobes in the same tube.[4]

In twenty-six cases, no medicament was applied to the dentin, these cases being used as controls. In the other cases, various medicaments were applied to the cavity on a sterile cotton pellet, which was left in for three minutes. At the end of this time, the cavity was wiped dry with sterile cotton and a circular pellet of sterile baseplate guttapercha was placed in the bottom of the cavity.

The stopping and carrying instruments were, of course, previously sterilized in the open flame. Over the guttapercha was placed a quick-setting zinc oxide and eugenol cement:

Powder	Gm.	Liquid	Cc.
Zinc oxide (regeant)	70		
Zinc stearate	8	Eugenol	88
W. W. rosin (U.S.P.)	20	Olive oil	12
Benzoic acid	2		

(Grossman[5] has shown that the combination of guttapercha and zinc oxide and eugenol cement will hermetically seal the dentin against penetration of saliva. In addition, the fact that guttapercha is inert and easily removed enhanced its value for this experiment.)

The patient was then dismissed for a period of from one to two weeks. In several cases, the cement broke away between visits and the underlying guttapercha was exposed. These cases were discarded. At the second visit, the tooth was again placed under the dam and the temporary filling removed. Of course, the same aseptic conditions were maintained as at the previous sitting. Shavings from the dentin were then again cultured and results noted.

The cavities were grouped under three general headings: (1) shallow, (2) medium and (3) deep. By shallow, that type of cavity is meant which can just be hooked by an explorer and in which the dentin has not yet been involved to any great extent roentgenographically (defective pits and fissures in the majority of cases). Medium cavities were those in which the dentin was involved by the carious process, but in which there was still a layer of healthy dentin between the floor of the cavity (after preparation) and the pulp. In these cases, evidence of caries in the dentin could be seen roentgenographically. In the deep group were placed those cavities in which little dentin remained after the

cavity preparation and also those in which there was a possibility of pulp involvement. In none of these cases could the dentin at the floor of the cavity be classified as "healthy."

All cultures were incubated for from seventy-two to ninety-six hours and then examined microscopically. Since this study was not concerned with the bacteriology of dental caries, it was deemed necessary to record only the presence or absence of bacteria.

An attempt was made to check every step of the procedure for a break in the chain of asepsis. 1. A piece of sterile paper having an area approximating that

Fig. 1.—Comparative effectiveness of the medicaments tested in the sterilization of dentin.

of an occlusal cavity in a molar was placed in a sterile petri dish. As soon as work was begun on the cavity, the lid of the petri dish was removed and the paper exposed to the air. When the cavity was sealed, the paper was picked up with sterile pliers and dropped into a tube of culture medium and incubated. This acted to determine whether a cavity could be contaminated merely by exposure to the air. The culture showed no growth. 2. A piece of guttapercha was softened in the flame, placed in a cavity, removed and dropped into a tube of culture medium, which also, after incubation, showed no growth. 3. The thought also occurred that perhaps bacteria were being incorporated in the mix of zinc oxide and eugenol cement. As a result, a mix was made on a slab that had been previously wiped with alcohol and chloroform, according to the usual procedure, allowed to set and then cultured. Again

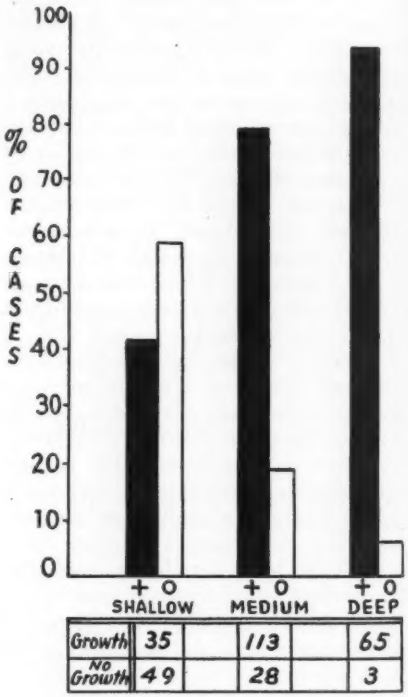

Fig. 2.—Bacteriologic status of the dentin after cavity preparation but before medication.

a negative result was obtained. 4. There was no need to check the sterility of burs and excavators after dipping in the molten metal sterilizer.

RESULTS

The medicaments tested were: (1) pure liquid phenol; (2) 50 per cent

phenol in ethyl alcohol; (3) 50 per cent thymol in ethyl alcohol; (4) Howe's ammoniacal silver nitrate; (5) 95 per cent ethyl alcohol; (6) isotonic solution of iodine; (7) 10 per cent aqueous solution of iodine; (8) Morson's kreosote, and (9) physiologic solution of sodium chloride, as a control. (Results of tests with 70 per cent ethyl alcohol will be reported in a subsequent paper.)

Table 1 shows the results obtained. In the control group (no sterilization), thirty-two cases were observed, of which twenty-four were originally positive and eight negative. On examination, at the second visit, eighteen positive and eight negative results were obtained. Six cases were either discarded or not further available. For all intents and purposes, there was no appreciable change bacteriologically.

The best results were obtained with beechwood creosote. Thirty-eight cases were tested. Of nine small cavities, six were originally positive and three were negative. On the second culture, five negative and only one positive returned. In the medium class, sixteen cavities were cultured, fourteen of which were positive and two negative. The second culture revealed only one positive and twelve negative, showing that ten cavities were actually sterilized. Three cases were discarded owing to breakage of the temporary filling in between visits. Of thirteen large cavities tested, ten were positive and three negative. The check culture on the second visit gave three positive and six negative. Four other cavities were discarded owing to leakage. Thus, of a total of thirty positive and eight negative results on the first culture, the second culture revealed only five positive and twenty-three negative; ten cases being either discarded or not available. This represents a jump from approximately 18 per cent originally negative cavities to 86 per cent, an increase of 68 per cent in sterility. (Table 2.)

Phenol 50 per cent in ethyl alcohol was disappointing as a sterilizing agent. Twenty cases were tested; four small, ten medium and six large. Sixteen of these were originally positive, four negative. Thirteen of these were found positive and three of these were discarded. Three patients failed to return. On the second examination, only four cultures

TABLE 1.

Before Treatment													After Treatment								
Shallow		Medium		Deep		Total		*	corrected Total			Shallow		Medium		Deep		Total		
+	o	+	o	+	o	+	o	X	+	o		+	o	+	o	+	o	+	o	
6	6	8	2	10	0	24	8	6	20	6	No Sterilization	6	4	5	3	7	1	18	8
1	3	12	1	4	0	17	4	4	13	4	Physiologic Saline Solution	1	3	7	4	1	1	9	8
3	4	4	1	2	0	9	5	3	9	2	95% Ethyl Alcohol	4	2	4	0	1	0	9	2
1	3	9	1	6	0	16	4	6	11	3	50% Phenol in Ethyl Alcohol	0	2	7	2	3	0	10	4
2	4	18	6	4	0	24	10	5	22	7	Pure Liquid Phenol	3	3	5	15	1	2	9	20
1	3	12	1	5	0	18	4	4	14	4	50% Thymol in Ethyl Alcohol	0	2	9	4	2	1	11	7
2	2	9	2	6	0	17	4	3	14	4	Isotonic Solution of Iodin	1	2	5	6	4	0	10	8
2	2	14	3	5	0	21	5	3	18	5	10% Aqueous Solution Iodin	2	2	2	13	1	3	5	18
0	3	11	5	10	0	21	8	4	20	5	Howe's Ammoniacal Silver Nitrate	1	1	11	4	6	2	18	7
6	3	14	2	10	3	30	8	10	24	5	Morson's "Kreosote"	1	5	1	12	3	6	5	23

*X = Number of cavities that were discarded due to leakage of the seal plus the number of cases that failed to return.

were negative. Thus, the second group of cultures actually revealed only one more negative than the first, indicating no sterilizing value.

Perhaps just as disappointing were the results obtained with 50 per cent thymol in ethyl alcohol. In this group, twenty-two cavities were tested. Eighteen were positive originally. On the second visit, seven negative and eleven positive results were obtained. Two of the original cavities were discarded and two patients failed to return. Thus, sterility was obtained in only three cases, while at least eleven remained in the same positive status.

TABLE 2.

	Before Treatment		After Treatment	
	% +	% 0	% +	% 0
No Sterilization	77	23	69	31
Physiologic Saline Solution	78	22	53	47
95% Ethyl Alcohol	81	19	81	19
50% Phenol Alcohol	78	22	71	29
Pure Liquid Phenol	77	23	31	69
50% Thymol in Alcohol	78	22	61	39
Isotonic Iodin	78	22	56	44
10% Aqueous Solution of Iodin	79	21	22	78
Howe's Ammoniacal Silver Nitrate	80	20	72	28
Morson's "Kreosote"	82	18	14	86

With isotonic iodine (about 0.3 per cent iodine), the results were indifferent. Twenty-one were treated in this group. Seventeen were positive and four negative at the first culture. On subsequent culture, ten positive and eight negative results were obtained; three cultures being discarded because of leakage. Thus, the second culture revealed a reduction in the positive cultures from 80 to 55 per cent.

When the concentration of iodine was raised to 10 per cent, a marked improvement in sterilizing ability was noted. Twenty-six cases were tested in this group, twenty-one of which were originally positive and five negative. On the second culture, eight positive and eighteen negative results were obtained. Three of the eight positive were discarded owing to leakage. Thus, a jump was detected here in sterility from 21 to 78 per cent, which is significant.

Howe's ammoniacal silver nitrate afforded some surprising results. Twenty-nine cases were tested. Twenty-one were positive and eight negative. On the second culture, there were twenty positive and only seven negative results; two cases failing to appear. Two of the twenty positive were discarded. Here, we actually had only two more negative on the second culture than on the first; which indicated almost no sterilizing value with this medicament.

Physiologic solution of sodium chloride was added to this list merely for a control, in place of distilled water. The results showed that it had some sterilizing value but were not consistent enough to be significant. Of the twenty-one cases under observation, seventeen were positive in the beginning and four were negative. On the second culture, there were nine positive and eight negative cultures, four patients failing to appear. This represents an increase in sterility from 19 to 47 per cent. Although we would hesitate to recommend a saline solution as a sterilizing agent, the results are better than with some of the more popular medicaments.

In the 95 per cent ethyl alcohol group, only fourteen cases were tested, nine being positive and five negative. When the nine cases proved positive, it was believed that there was no point to continuing with this solvent since it was obvious that it has no germicidal action whatsoever.

Pure liquid phenol yielded good results. A total of thirty-four cases were

tested. Twenty-four were originally positive and ten negative. On checking, there were nine positive and twenty negative results, five patients failing to return. Thus, the original 23 per cent negative total was increased to 69 per cent, a creditable showing.

Arranging the drugs in order of their efficiency, we get the following: (1) Morson's kreosote; (2) 10 per cent aqueous solution of iodine; (3) pure liquid phenol; (4) physiologic solution of sodium chloride; (5) isotonic solution of iodine; (6) 50 per cent thymol in ethyl alcohol; (7) 50 per cent phenol in ethyl alcohol; (8) Howe's ammoniacal silver nitrate, and (9) 95 per cent ethyl alcohol. (Fig. 1.)

COMMENT

Before considering the results obtained with sterilizing agents, it might not be amiss to reemphasize a significant fact that has come to light from this work. In a previous publication,[6] it was pointed out that the larger the cavity, the less chance the operator has of removing bacteria from the dentin mechanically. This evidence emphasizes the necessity for cavity sterilization prior to a restoration. Since then, additional data have been collected which tend to enhance the conclusions drawn at that time. (Fig. 2.)

In a small percentage of cases, cavities that yielded negative cultures on the first visit gave positive results on the second. This may be attributed to two main facts:

1. There are an insufficient number of bacteria in the first sample, with inability of a small number of bacteria to propagate themselves in a comparatively large volume of medium. 2. There possibly is contamination of the cultured medium in taking the second culture.

On the other hand, in a number of cases, the second culture yielded negative results as compared to an originally positive culture. When no medicaments were introduced or the medicaments had a questionable antiseptic action (as when physiologic solution of sodium chloride was used), a second negative culture following a first positive culture might be due to (1) an insufficient number of bacteria in the second sample or (2) contamination of the first culture.

In the experiments in which a large increase in negative cultures was found, one could rightfully attribute these findings in part, at least, to the action of the drugs.

It is not surprising that the results with 50 per cent phenol in ethyl alcohol and 50 per cent thymol in ethyl alcohol are approximately the same. Both solutes are relatively insoluble in water. Both are caustics and both are dissoluble in alcohol. When the last fact was noted, it was decided to test the germicidal value of the solvent itself. Only fourteen cases were tested, but these were sufficient to show that, as a bactericidal agent, 95 per cent ethyl alcohol has no value whatsoever. When results obtained with pure liquid phenol were at least 40 per cent better than those with 50 per cent phenol in ethyl alcohol, it was believed that no further proof was needed to show that the use of ethyl alcohol as a solvent decreased the bactericidal action of a drug, at least in cavity sterilization. As a matter of fact, Appleton[7] expressly stated that "every addition of ethyl or methyl alcohol to an aqueous solution of phenol or of formic aldehyde decreases its disinfectant action."

Prinz,[8] who recommended a 50 per cent solution of thymol in alcohol,[9] specifically stated that "solutions of phenol in concentrated alcohol or in fatty oils are comparatively worthless." However, in the same paragraph, he also said that "certain other antiseptics, as phenol, lysol, and thymol, act more forcefully when dissolved in 50 per cent alcohol than when an equal quantity is dissolved in water." However, *res ipsa*

loquitur, the bacteriologic findings point to the comparative worthlessness of solutions made with ethyl alcohol as the vehicle.

With our present knowledge of the histology of carious dentin, the failure of alcoholic solutions and of silver nitrate to induce sterility can be explained by the fact that all are severe caustics, acting as germicides or antiseptics by virtue of the fact that they coagulate the albumin of protoplasm. It is quite possible, then, that the surface of the dentin may be sterilized while bacteria continue to lurk in the dentinal tubules below the surface. The coagulation of the albumin in the tubules by the caustics makes these agents self-limiting. This very fact causes them to lose their appeal as dentin sterilizing agents. How, then, does silver nitrate act so successfully from the clinical standpoint? This may be answered, it is believed, as follows:

Fish[10] has shown that at the inception of the carious process, the pulp starts to lay down a barrier of secondary dentin for protection against poisonous products of bacterial metabolism. When applied to the cavity, silver nitrate coagulates the protoplasm on the surface, plugging up the tubules just as a cork closes the neck of a bottle. Thus, the bacteria which are still alive in the tubules are trapped on the pulpal side by secondary dentin and on the outside by coagulated albumin.*

From histologic studies, it is known that bacteria penetrate the dentin beyond the point of liquefaction of the dentin. As Hopewell-Smith[11] pointed out:

> After the micro-organisms have once entered the tubules, they easily pass along their lumina. The canals then become choked with micrococci, gradually enlarge and give way in numerous places.

Appleton[6] says:

> In any tooth in which dental caries has involved the dentin, the pulp must be regarded as at least potentially infected. The fact that the bacteria have always penetrated further into the dentinal tubules than is indicated by any gross change in the dentin necessitates, in cavity preparation, the sterilizing of the cavity wall, before introducing the filling material.

It is a well-known pharmacologic law that an antiseptic is effective only in proportion to the contact that it makes with the bacteria. In other words, an antiseptic obviously cannot be effective unless it actually comes in contact with the bacteria. Since it has been shown that alcohol, by virtue of its coagulating action and in other ways, decreases the antiseptic action of a drug, it appeared that aqueous solutions would be more efficient as dentin-sterilizing agents. The results with pure phenol corroborated this. With this in mind, it was felt that perhaps an isotonic solution of iodine might be an efficient agent. The indifferent results that followed experimentation with this agent suggested the possibility that the low concentration of iodine in this solution (about 0.3 per cent) and not the iodine itself might be at fault.

Recent investigations by Salle, McOmie and Shechmeister,[12] working on embryonic chick's heart tissue, have brought to light the fact that iodine, which is one of the oldest germicides known, is the most nearly ideal of all of the compounds tested from the standpoint of low toxicity plus germicidal action. It is usually employed as a tincture, which is irritating, owing to the presence of alcohol. In aqueous solution, however, the iodine is non-irritating and relatively non-toxic to tissue.

It was decided, then, to use an aqueous solution of iodine at a higher concentration and a 10 per cent solution was arbitrarily tried. The results were gratifying, although not quite so good as those obtained with Morson's kreosote.

As far as discoloration of the tooth

*Results of clinical studies on the treatment of carious cavities with Howe's ammoniacal silver nitrate are in press.

with iodine is concerned, it may be pointed out that a reddish brown discoloration is produced as soon as the iodine touches the dentin. Carious dentin stains much more intensely than normal dentin. The stain, however, is short-lived. It disappears completely in a short time owing to a chemical reaction between the dentin itself and the iodine, and not to removal of the stain, which might occur through a circulation in the dentin. Corroboration of this fact was obtained when it was noted that several extracted teeth, treated and sealed in exactly the same manner as those in the mouth, were destained.

With Morson's kreosote, the results were better than with any other medicament tested. C. E. Kells[18] was long an advocate of this medicament. However, I have been unable to find more than a few practitioners who have employed this drug in their practices. In his book, Kells admitted that he had no scientific basis for using Morson's kreosote, but had acquired the habit of usage from his father. He used it for nearly every dental purpose and reported favorably on his clinical results.

Since becoming acquainted with this product, I and several of my co-workers have been using Morson's kreosote for various purposes other than cavity sterilization. As a sedative, it has given as much satisfaction as oil of cloves. When mixed with zinc oxide powder, mentioned earlier in this paper, it makes an excellent pulp-capping material or subcement base. It is applied over an exposed pulp without heat and can be pushed gently into place with a minimum of pressure. It is highly adhesive and sets rapidly in the mouth. Observations on this material will be reported at a later time.

An attempt was made to find out whether there is any actual difference between Morson's kreosote and the ordinary U.S.P. beechwood creosote. We have only the word of the manufacturer, who states that this product differs by virtue of the fact that it is manufactured under a special maturing process which tends to eliminate certain irritating and undesirable properties from the creosote. Also, the manufacturer claims that the creosote is extracted from special trees grown in England. H. L. Hansen stated that the Council on Dental Therapeutics of the A.D.A. had no data on Morson's kreosote and, as far as he had been able to ascertain, it did not differ in essential respects from U.S.P. beechwood creosote. The editor of the *British Dental Journal* likewise reported a lack of data on this product. U.S.P. creosote is being tested, but I am unable, at this time, to report on its efficiency as a dentin sterilizing agent owing to insufficiency of data.

The majority of the medicaments do not afford sterility in every case and some sterilize in few cases; yet certain practitioners have success with these agents. The reasons may be several: 1. There can be no question that removal of all carious material is of paramount importance. By this operation, analogous to surgical removal of diseased tissue anywhere else in the body, we are removing a tremendous number of bacteria and automatically giving the tissue a better chance to combat the carious process. 2. The resistance of the tissues, i.e. the ability of the pulp to lay down a protecting layer of secondary dentin, must be taken into consideration. Age, general health, diet, heredity and various other conditions materially influence this ability. Equally important, perhaps, is the rapidity of the carious process; that is, the acuteness or chronicity of the disease. All of us have, at some time or another, been surprised to see how large a cavity can become in a few months. In other cases, pits that were detected three or four years previously have become no larger.

With this in mind, it becomes under-

standable that a practitioner may not sterilize cavities at all and yet, to all appearances, the particular filling looks well. Perhaps, in this case, every trace of carious material was removed and merely sealing the cavity hermetically with a good filling plus secondary dentin formation by the pulp was sufficient to take care of any remaining bacteria. On the other hand, it has also been our experience to find recurrence of caries under fillings that have been inserted by good operators, men who conscientiously and meticulously remove all carious material and who endeavor to sterilize the cavities prior to insertion of the filling. It is in these cases that we can blame the so-called sterilizing agent for the failure, and because of these cases, it is imperative that we use an agent that is known to sterilize in every case.

CONCLUSIONS

1. The dentin is a difficult substance to penetrate and hence difficult to sterilize.

2. Protoplasm caustics such as silver nitrate and 50 per cent thymol in ethyl alcohol do not sterilize dentin under conditions of the test because they are self-limiting and hence do not come in contact with the bacteria in the dentinal tubules. Their antiseptic action can be attributed to a "walling off" of bacteria.

3. Drugs dissolved in 95 per cent ethyl alcohol might well be discontinued for use in cavities. Aside from inhibiting the action of drugs bacteriologically, alcohol also sets up a distinct irritation to the pulp since it makes the solution hypertonic. All alcoholic solutions are apt to be painful.

4. The best bacteriologic results were obtained with Morson's kreosote and with a 10 per cent aqueous solution of iodine. Good results were also obtained with pure liquid phenol.

5. Morson's kreosote mixed with a zinc oxide powder is being tested as a subcement base for pulp capping and other purposes.

6. Bacteriologic studies are convincing evidence that all cavities should be opened and filled as soon as detected.

BIBLIOGRAPHY

1. MILLER, W. D.: Comparative Rapidity with Which Different Antiseptics Penetrate Decalcified Dentine; or What Antiseptic Should Be Used for Sterilizing Cavities Before Filling? *D. Cosmos*, 33:337, May 1891.

2. BARKER, J. N.: Sterilization of Dentin. *Australian J. Dent.*, 29:156, May 1, 1935.

3. APPLETON, J. L. T.: Tests on Efficiency of Molten Metal Sterilizer. Unpublished data.

4. APPLETON, J. L. T., and BRYANT, C. K.: Laboratory Guide in Bacteriology. Philadelphia: Lea & Febiger, 1928, p. 38.

5. GROSSMAN, L. I.: Study of Temporary Fillings as Hermetic Sealing Agents. *J. D. Res.*, 18:67, February 1939.

6. SELTZER, SAMUEL: Bacteriologic Status of Dentin After Cavity Preparation. J.A.D.A., 27:1799, November 1940.

7. *Idem:* Bacterial Infection. Ed. 2. Philadelphia: Lea & Febiger, 1933, pp. 50:443.

8. *Idem:* Dental Materia Medica and Therapeutics. Ed. 2. Philadelphia: Lea & Febiger, 1912, p. 109.

9. PRINZ, HERMANN: Diseases of Soft Structures of Teeth and Their Treatment. Philadelphia: Lea & Febiger, 1928, p. 109.

10. FISH, E. W., JR.; Physiology of Dentine and Its Reaction to Injury and Disease. *British D. J.*, 14:593, June 1, 1928.

11. HOPEWELL-SMITH, ARTHUR: Normal and Pathological Histology of Mouth I. Vol. II. Ed. 2. Philadelphia: P. Blakiston's Sons & Co., 1918, pp. 102 and 132.

12. SALLE, A. J.; MCOMIE, W. A., and SCHECHMEISTER, I. L.: New Method for Evaluation of Germicidal Substances. *J. Bact.*, 34:267, September 1937.

13. KELLS, C. E.: Three Score Years and Nine. Published by author, 1926, pp. 72, 248.

14. COOLIDGE, E. D.: Treatment of Deep Dental Caries. *Illinois D. J.*, 1:363, May 1932.

15. HOWE, P. R.: Method of Sterilizing and at Same Time Impregnating with Metal, Affected Dentinal Tissue. *D. Cosmos*, 59:891, September 1917.

16. PRIME, J. M.: Controlling Dental Caries. J.A.D.A., 24:1950, December 1937.

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THE ETRUSCAN CAST "GOLD" CROWNS ILLUSTRATED BY GUERINI

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IF one is inclined not to accept facts at their face value and is interested in doing a little investigating for himself, let him undertake the study of dental history. For romance and intrigue, there is little to compare with it.

The trouble is, however, that when one attempts a historical study, he generally accepts that which appears in the histories as gospel, and continues to repeat these statements indefinitely. I myself have been guilty of doing this very thing until I was able to find some evidence to the contrary.

In Guerini's "History of Dentistry," page 70 (Figs. 13 and 14), are photographs bearing the legend "Tooth crowns found in an Etruscan tomb of the ancient Vitulonia (Archaeological Museum of Florence). The enamel-capsules of the teeth (four molars and one canine) are perfectly well preserved, whilst the ivory has entirely disappeared." Similar crowns are to be found among others uncovered in ancient tombs. It is this fact more than any other that has tended to confuse the reader. The other misleading reference is to the "disappearance of the ivory," and from it one would conclude that an artificial crown of some type had formerly been placed on a carved ivory root. These crowns, I was informed, were of cast gold.

That this idea is a prevalent one, there is no doubt; for it has been repeated by such well-known historians as the late L. Parmly Brown. On page 958, *Dental Cosmos*, 1934, he wrote: "Some cast gold tooth caps or crowns. . . ." However, he too must have questioned the statement, for he ends the paragraph by saying: "But we have no definite record of any other independent gold caps until about 1593." While here in 1926, Guerini gave me the impression that they were cast gold crowns.

Upon making several inquiries of the Archaeological Museum in Florence, a reply was finally received containing the following statement: "But there exists

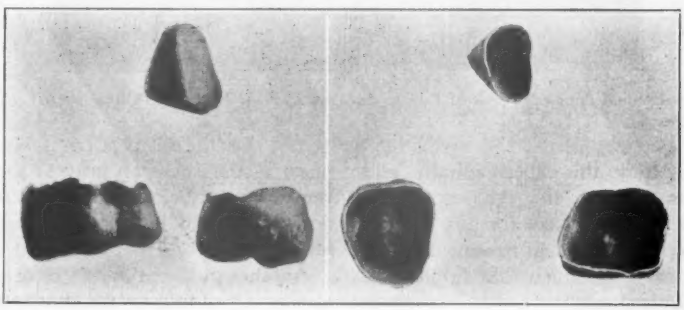

Fig. 1.—Supposed "cast gold crowns." (From Guerini's "History of Dentistry.")

no trace of such specimens in our topographical Etrurian Museum and I don't know from where Guerini has this information."

Learning that there were no such crowns in the Museum only helped to increase the mystery. What, then, could they be? Could the dentin and cementum have been dissolved by some chemical that would not at the same time break down the enamel? Those active in histologic research stated that it was impossible to do this experimentally, and if it were possible, the evidence would not be a sufficient basis for any conclusions. We therefore had to wait until we came across a specimen that fulfilled all of the requirements.

Several years ago, Henry W. Gillett obtained from an Indian grave in the Southwest a collection of Pueblo molar teeth, which are now in the possession of Dr. Bodecker, of Columbia University, their estimated age being about 900 years. During these years, the dentin and cementum disintegrated so that but a small round ball remained within the enamel cap. This small ball was easily lifted out, leaving the enamel cap intact, as shown in Figure 1. A comparison with the illustration in Guerini's history shows the internal structures to be alike.

Undoubtedly, a similar reaction took place in the Etruscan graves; therefore, the supposedly cast gold crowns on page 70 turn out to be but enamel caps of human teeth and not cast gold crowns.

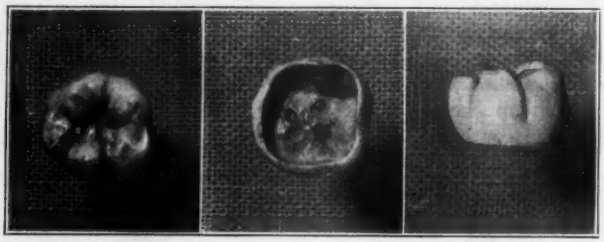

Fig. 2.—Enamel caps of Teeth of Pueblo Indians of New Mexico. (Circa 1040.) (Courtesy Dr. Bodecker.)

Another of the mysteries of dental history is solved and we see illustrated how easily, when facts are not clearly presented or stated, misinterpretations arise and are continued in dental history.

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DENTAL STRUCTURES REMAINING IN 1,948 EDENTULOUS JAWS: A STATISTICAL STUDY

By DONALD T. WAGGENER,* D.D.S., and LOUIE T. AUSTIN, D.D.S., Rochester, Minn.

EDENTULOUS jaws were examined clinically and roentgenographically in a series of consecutive extraction cases. This series gives the average result of service in dental extraction, including the results of the most conscientious as well as the most negligent dentists. There were 1,380 patients in this series who had had the teeth removed from one or both jaws. These patients, if each had the normal number of teeth in each jaw, had had a total of 31,125 extractions. There were 748 patients who were edentulous only in the upper jaw, and 15.5 per cent had upper roots remaining. Sixty-four patients were edentulous in the lower jaw only, and 15.6 per cent had lower roots remaining. Five hundred and sixty-eight patients were edentulous in both jaws, and 27.4 per cent had roots remaining. The total number of roots was 410; which indicates a failure of 1.3 per cent in the 31,125 teeth extracted.

The data concerning the dental structures not removed from the jaws of patients from which the teeth had been extracted are recorded in Tables 1 and 2. Table 1 gives the situation in which roots were found in the dental arches and the percentage of failure of complete removal for each individual tooth. From the known number of each of the various teeth that have been extracted and the number and location of roots remaining in each jaw, the percentage of efficiency in removal of individual teeth can be determined. The complete re-

TABLE 1.—NUMBER AND SITUATION OF ROOTS REMAINING IN EDENTULOUS JAWS AS REVEALED BY ROUTINE ROENTGENOGRAMS AFTER REMOVAL OF 31,125 TEETH

	Left Side								Right Side								Total
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	
Upper Percentage failure	1.38	3.64	1.82	1.57	1.89	0.75	0.60	0.37	0.75	0.45	0.68	2.2	1.89	1.89	2.81	2.06	1.46
Roots remaining	18	48	24	21	25	10	8	5	1	6	9	29	25	25	37	27	318
Lower Percentage failure	1.27	0.15	1.10	0.79	1.26	0.63	0.79	0.63	0.47	0.15	0.94	1.42	0.94	1.26	0.79	1.91	0.86
Roots remaining	8	1	7	5	8	4	5	4	3	1	6	9	6	8	5	12	92

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From the Section on Dental Surgery, Mayo Clinic.

moval of the upper teeth apparently was more difficult than the complete removal of the lower teeth. There was a failure of 1.46 per cent in removal of upper teeth as compared with a failure of 0.86 per cent in removal of lower teeth.

Table 2 gives the location of the forty-three remaining unerupted teeth, other than supernumerary teeth, in the same series of jaws which were otherwise edentulous. It will be noted that unerupted upper third molars are left in the jaw most frequently. Perhaps this is because they cause painful symptoms less frequently than do unerupted lower third molars; therefore, their presence may be more easily overlooked until that area is roentgenographed. Since about one of every thirty-two patients in this series had an unerupted tooth other than a supernumerary tooth remaining, the importance of taking full-mouth roentgenograms before extraction should be emphasized.

Three hundred and twenty-nine patients had roots, unerupted teeth of the permanent dentition or supernumerary teeth which had not been removed; 23.8 per cent of the entire series of 1,380 patients. Eleven cysts were discovered, six of which were median anterior maxillary cysts, and five were cysts associated with dental structures. There were also eight supernumerary teeth remaining, seven of which were located near the midline of the maxillae.

Table 3 records sex and age distribution in this series. It is interesting to note that the average age at which the women became edentulous in one or both jaws is nearly three years less than the corresponding age for the men. The mean age was 44 years.

TABLE 2.—NUMBER AND SITUATION OF UNERUPTED TEETH (OTHER THAN SUPERNUMERARY TEETH) IN 1,948 OTHERWISE EDENTULOUS JAWS AS REVEALED BY ROUTINE ROENTGENOGRAMS

	Left Side								Right Side								Total
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8	
Upper teeth remaining	9	1	0	1	0	2	0	0	0	0	7	0	1	0	0	9	30
Lower teeth remaining	4	0	1	0	0	3	0	0	0	0	1	0	0	0	0	4	13

TABLE 3.—SEX AND AGE DISTRIBUTION

Age	Total		Men		Women	
Years	Number	Per Cent	Number	Per Cent	Number	Per Cent
10-19	9	0.7	2	0.3	7	1.0
20-29	126	9.1	48	7.2	78	10.9
30-39	363	26.3	158	23.8	205	28.6
40-49	489	35.4	231	34.9	258	36.0
50-59	295	21.4	164	24.7	131	18.3
60-69	91	6.6	57	8.6	34	4.7
70 and more	7	0.5	3	0.5	4	0.5
Total	1,380	100	663	100	717	100
Mean age	44.0 years		45.4 years		42.6 years	

TABLE 4.—AGES AT TIME OF EXTRACTION

Age	Upper		Lower		Both*	
Years	Number	Per Cent	Number	Per Cent	Number	Per Cent
10-19	5	0.7	0	0	3	0.6
20-29	78	10.4	2	3.1	39	7.6
30-39	222	29.7	12	18.8	108	21.1
40-49	256	34.2	19	29.7	194	37.8
50-59	150	20.1	25	39.0	117	22.9
60-69	33	4.4	5	7.8	50	9.8
70 and more	4	0.5	1	1.6	1	0.2
Total	748	100	64	100	512	100
Mean age	42.8 years		48.4 years		54.5 years	

*In fifty-six cases in which patients were edentulous in both jaws, enough time elapsed between complete removal of the teeth from each jaw to cause the two jaws to fall into different age groups. These cases were not included in this table.

Table 4 gives the ages of the patients at the time of extraction. The upper teeth were removed at an average age five and six-tenths years less than the age at which the lower teeth were removed. When only the upper teeth or both the upper and the lower teeth were removed, the greatest percentage of the patients were in their forties. When only the lower teeth were removed, the greatest percentage of the patients were in their fifties.

Cook[1] reported a similar series of 500 patients studied approximately ten years before the present series. Twenty-six per cent of the patients in Cook's series who were edentulous only in the upper jaw had roots remaining. Twenty per cent of those who were edentulous only in the lower jaw had roots remaining. Twenty-nine per cent of those who were edentulous in both jaws had roots remaining. These percentages are significantly higher than those of the present series only in regard to the extraction of teeth from the upper jaw.

These data emphasize the importance of making sure that roots of extracted teeth are removed completely and the value of roentgenographic examination in establishing this fact. Such a service is especially important to the patient who has been entrusted to the dentist for the elimination of focal infection of dental source.

BIBLIOGRAPHY

1. COOK, T. J.: Statistics Obtained by Clinical and Roentgenographic Examinations of 500 Edentulous and Partially Edentulous Mouths. *D. Cosmos*, 69:349-351, April 1927.

DENTAL CARIES IN PREGNANCY

By JOSEPH D. JAMES, M.D., Springfield, Mo.

THERE was accepted in the dim past the tradition that pregnancy is the precursor of dental caries, a tradition that was popularized in the legend that for every baby, a tooth! This is still an axiom among a large percentage of the laity and some members of the dental and the medical profession. It is only recently that the truth of this so-called axiom has been questioned. It is unnecessary to state that proper dental care is needed for every individual and that care is especially necessary during pregnancy in consideration of the great harm that can result from improper care or neglect of the teeth during that time.

It has long been recognized that there is a direct relation between focal infection and the late toxemias of pregnancy, pyelitis, improper nutrition of the mother and the growing fetus and inadequate lactation. The ravages of caries are greater during pregnancy and its advance is more rapid and painful. In this paper, it seems proper, though digressing, to discuss briefly the embryology of the teeth and the pathology of dental caries and to refer to some of the various theories of the cause of decay.

It is a well-established fact that the structure of the teeth is laid down before the fifth month of intrauterine life, and proper measures for their welfare are an obstetrical and a maternal problem; at least, as far as the deciduous teeth are concerned; and since calcification of the permanent teeth takes place largely after birth, the responsibility then becomes that of the pediatrician.[1, 2, 3, 4, 5]

Faulty tooth structure is first noted in the deciduous and the early permanent

Read before the Springfield, Missouri, District Dental Society.

teeth. Dental caries is primarily a disease of childhood and adolescence. The future health of the teeth depends, to a large extent, on heredity, the nutrition of the expectant mother and the supervision of the nutrition of the child well into adolescence.[6, 7, 8, 9, 10]

There are two important types of dental caries. One occurs in the teeth of the young, beginning in either pits or fissures in the crown of the tooth or at the contact point of approximating teeth, the ectodermal layer. These faults generally penetrate the enamel with only a sharply localized destruction of that tissue and spread widely in the dentin and the mesodermal layer and approach the pulp very early, with rapid demineralization of the teeth and digestion of the non-mineral elements. The other type occurs more frequently in the teeth of the adult and the aged and commonly attacks the crown or the exposed root surfaces, producing a large aperture on the surface and more slowly invading the deeper structures.

Bacterial invasion is a factor in hastening the destruction of the teeth in both types. The state of health of the body seems to make little or no difference in the incidence of caries once the dental structure has been laid down, during intrauterine life, and the permanent teeth are fully erupted. The statement that constitutional disease and pregnancy are factors in dental caries is not warranted and lacks confirmation by recent investigators. On the other hand, as has been stated, the acute infectious diseases of early childhood can affect the structure of the growing tooth.[2, 6, 8, 9]

Oral hygiene has been "touted" as almost a panacea for the ills of the teeth and as a means for prevention of dental decay; and one is almost inclined to give credence to the statements to that effect in consideration of the propaganda regarding the different types of toothbrushes, toothpowders and toothpastes, each supposed to have some special merit. It has gone so far that one can almost visualize a different type of brush and paste for use after each meal and at bedtime in order that one may lie down to sleep with the assurance that "a clean tooth never decays." This sounds plausible, but it is not the truth and leads to a false sense of security.[5, 6, 7]

That oral hygiene is of no value is not the point. Its value from an esthetic standpoint is high; and if acid plaques are a cause of dental caries, it seems possible that early removal by brushing would be of some value. All of us are aware that some of the best teeth seen are the least brushed and some of the worst seen are the most brushed. It seems possible that those with the most brushed teeth are the poorest nourished.[9, 11]

It seems that the enamel of the tooth may be destroyed by the action of weak acids[6, 12] with later demineralization of the inorganic portion and liquefaction of the organic constituents. This lesion, together with bacterial invasion, leads to complete destruction of the affected tooth and to local septic conditions. This, with the theory that plaque formation is a possible cause of dental decay, in a way summarizes the leading theories regarding the mechanism of dental caries, but throws little light on the etiology. Some rather recent investigations would tend to discredit at least the chemical theory as far as acids are concerned, since, in the hyperacidity and vomiting of pregnancy, in which the mouth is at times highly acid, the teeth are unaffected; rather, seeming freer from progressive caries. Recent investigators have found no greater incidence of caries in the pregnant than in the non-pregnant of the same age. It appears that pregnancy has a beneficial effect in preventing dental caries, but a preexisting caries may be aggravated.[3, 5, 6, 11, 12, 13]

DIET

All investigators accept the opinion that proper food in necessary amounts

during pregnancy, and especially early in pregnancy, is essential to the normal development of the deciduous as well as the permanent teeth, since the foundation of the tooth structure starts in the seventh week in the human embryo, calcification begins as early as the seventeenth week and the crowns of the teeth are, for the most part, formed before birth.[2, 4, 5, 12]

The nutritional needs of the mother and of the fetus are identical. The diet during gestation should be sufficient to satisfy maternal requirements and to assure an adequate amount of minerals and vitamins, which should be obtained in natural foods instead of the various laboratory made compounds that are being advertised to the laity and the dental and medical professions.[2, 4, 14] However, proper diet is not absolutely essential to fetal development since embryonic tissue possesses a peculiar growth impulse. It is able to obtain through the placental circulation nutrient materials present in the blood plasma of the mother even though the supply is small; and in a deficiency of the many constituents needed for growth, in the blood plasma of the mother, a sufficient supply can be obtained from that stored in the mother's tissues.[4, 5, 12, 15]

ENDOCRINOLOGY

It is well known that disturbances of the parathyroid, the thyroid and the hypophysis have a marked effect on the growth and development of the fetus. Overactivity of the parathyroids causes decalcification, the bones, in extreme cases, becoming soft, with resultant skeletal deformities, as seen in osteomalacia.[4]

The parathyroids are the most important organs as far as calcium metabolism is concerned. Removal of these glands disturbs the calcification of the dentin and causes hypoplasia of the enamel. Fleischman found many instances of hypoplasia of the enamel in patients with a history of convulsions, which frequently accompany tetany in early life. In experimental hyperparathyroidism, the effect of a single injection of parathyroid extract on calcification in the normal animal was studied by Schom and Haun, who found that the blood calcium rises above normal at first and then returns to normal. Histologic study showed that the dentin which formed during the rise was poorly calcified and that the dentin formed during the fall was excessively calcified. In a study in which multiple injections of parathyroid extract were administered, similar changes in the dentin were observed. In addition, the alveolar bone showed osteoclastic activity and fibrous changes in the bone marrow.[5, 16]

HYPOPHYSIS

The removal of the hypophysis causes, among other changes, a marked retardation of the eruption and disturbance of calcification, the structure appearing more dense than normally.[9, 16]

Almost all of the glands that exert characteristic effects on the teeth affect the calcification of the teeth. This is notably true in intrauterine and early life. Endocrine disturbances in children usually increase the severity of dental caries.[10, 16]

Vitamins A and C seem to have a much less direct influence on the nutrition of the teeth than does vitamin D. No doubt, all the vitamins have a part in the formation of tooth structure, but it appears in the light of recent investigation that A, C and D have a much greater influence than all others. In the main, it seems that a vitamin A and C deficiency tends to produce a hypoplastic condition of the enamel, and that, in a deficiency of vitamin C, the dentin as well as the enamel may be affected.[4, 6, 15] On the other hand, certain later investigators doubt whether hypoplasia of the enamel is a factor in dental caries.[6, 7, 9] A vitamin D deficiency is similar in ac-

tion on calcium metabolism to injections of parathyroid extract. The rôle of vitamin D seems to be the mobilization of the calcium and acceleration of its absorption from the intestinal tract. It is believed that it also acts to lower the level of the serum phosphate. A change in the normal calcium : phosphorus ratio will disturb calcification. The normal functioning parathyroids and vitamin D in proper amounts are the stabilizers of calcium-phosphorus metabolism. However, a vitamin D deficiency alone will not cause caries.[16, 17]

The clinical problem of preventing the effects of a vitamin D deficiency is not serious today. The result of administration of vitamin D in the form of cod liver oil, as a safe source of the vitamin, is well known. In fact, the problem is to safeguard the public against the effects of hypervitaminosis from the careless and too liberal use of the concentrates.[16]

CALCIUM

The calcium content of the blood serum is normally 9 to 11 mg. per hundred cubic centimeters and it is constant under normal conditions. It is regulated by the parathyroid hormone and vitamin D. Of the serum calcium, there are two distinct physiologic fractions, a diffusible and a non-diffusible. The diffusible, which is capable of passing through the capillary wall, is the physiologically active calcium fraction.[17]

It has been shown that during pregnancy, calcium and phosphorus are retained by the maternal organism. The amount retained is more than accounted for by fetal utilization and perhaps represents a reserve supply for emergencies. This calcium storage is particularly marked during the latter months of pregnancy and is associated with a tendency toward a diminished supply of the serum calcium at this time.[17]

During the early months of pregnancy, the average value of calcium is 10.6 mg. to 100 cc. of blood. During the early stage of labor, this is decreased to 9.6 mg. A marked decrease in the mother's blood calcium occurs during early lactation, and this is not accounted for in the mother's milk. The total calcium excreted at this time through the feces and the urine is greater than the total intake.[17]

CALCIFICATION OF TEETH

Bones are decalcified and recalcified, but teeth undergo nearly all of their calcification early in their life cycle. The teeth undergo only a limited degree of secondary calcification and are not subject to the withdrawal of calcium. Disturbances of calcium metabolism, therefore, influence only the growing teeth; that is, the teeth of the fetus, the infant and the growing child. The tooth that is fully formed, calcified and erupted is not influenced by systemic disturbances in calcification.[9] Mull, Bill and Kinney, after a study of a large series of patients, concluded that there was no direct relation between the teeth and serum calcium and inorganic phosphorus during pregnancy.[11] The question of withdrawal of calcium from the teeth is therefore sufficiently important to justify a brief review of the literature.[16]

Nearly 2,000 years ago, Galen considered the teeth bits of bone. In the sixteenth century, Eustachi disproved this theory, showing that the teeth were cutaneous appendages. Two hundred years later, with the rise of the science of physiology and the employment of experimental methods, John Hunter conducted experiments with madder showing that when it was fed there was a red coloration of only the tissues that were calcifying.[16] In the animals that were allowed to live weeks after madder was fed, the red coloration disappeared from the bone, but did not disappear from the dentin. Hunter therefore concluded that there is a vital difference between bone and dentin and that bone

can be resorbed and its calcium be withdrawn.[16]

In a later experiment, Geis[18] used trypan blue. If the stain was injected before the permanent teeth had begun to form, all of the enamel was stained blue. If the trypan blue was given soon after the enamel had begun to form, the teeth were white, except for a blue area in the cervical zone. If the injections were made after the enamel of the permanent teeth was fully formed, the enamel remained white and the pulp and the adjacent dentin were stained blue. Some of the experimental animals (dogs) were kept under observation for years. The blue of the teeth did not change, although the dye was gradually eliminated from all other tissue. Sections through such teeth showed the dye in both enamel and dentin.

Fish kept a pregnant dog on a diet very poor in calcium. At the beginning of the experiment, a tooth was extracted and the amount of calcium in it was determined. At the end of the experiment, the bones were soft and decalcified to such an extent that they were hardly visible in the roentgenogram, and could be cut with a knife. The teeth, however, showed the same density roentgenographically, and chemical analysis showed the calcium content of the teeth to be unaltered.[18]

Fish also found that there was a gradual slight increase in the calcification of the dentin with advancing age, but never a calcium loss. He came to the conclusion that it is not possible to modify the calcium content of the dentin, by parathyroidectomy, a calcium deficient diet, a condition of pregnancy or feeding of therapeutic or excessive doses of vitamin D. Nor did two dogs with extreme natural softening of the bones show any loss of calcium from the dentin.[18] Fish's experiment was paralleled by Albright, Aub and Bauer, with clinical and laboratory findings in seventeen patients with hyperparathyroidism and decalcification of the bones. They found that the teeth did not take part in the generalized decalcification. Unlike the bones, the teeth are not subject to calcium withdrawal.[18]

Mottling of the enamel, due to a large fluorine fraction in the drinking water, is endemic in certain parts of the world.[19] It appears that the highest incidence of mottling of enamel is seen in cases of enamel hypoplasia; also that the incidence of caries is no greater in mottled enamel than in normal enamel. The effect of ingestion of toxic amounts of fluorine on the teeth may be cited as evidence of a fixed tooth structure in the early life of the tooth.

Fluorine affects only the teeth the enamel of which is being formed. Its effects are present when the teeth erupt. In other words, one who has spent the first six or seven years of life where the water is high in fluorine is liable to have mottled enamel in the permanent teeth. On the other hand, the child who spends the same period of life where the water is fluorine free and later moves to an area where the water carries a toxic amount of fluorine will not have mottled enamel, since, at that age, all the enamel is completely calcified.[18] The third molars will be mottled only if the child has lived in the area during the time of their calcification, which is from the seventh to the tenth year.[20] The defective calcification or the mottling of these teeth cannot be changed by treatment.[18]

For the most part, the dental tissues are calcified during the formative stage, prior to eruption.[3] Only in the dentin and the cementum does calcification of newly formed matrix continue throughout life. During their development, the human teeth are very sensitive to variations in calcium supply and utilization, but this is not true of the erupted adult tooth. Calcium may be withdrawn or withheld from the enamel of the teeth during the early life of the teeth, by tissue metabolism. Bone may be decalci-

fied by osteoclastic resorption or by direct dissolution of the calcium salts, but no such process has ever been demonstrated in the developed human teeth.[16]

The Council on Dental Therapeutics recently published a comprehensive report entitled "Calcium and Phosphorus Compounds in Dentistry," in which its position against indiscriminate calcium therapy is shown to be well founded.

On the basis of our present knowledge, calcium therapy *per se* in dentistry is not indicated. When the individual condition seems to call for such therapy, treatment should be on the basis of medical and systemic needs[16] and becomes a medicodental problem. Aside from the fact that calcium therapy is not needed in the vast majority of cases, there is a possible danger in indiscriminate routine use of calcium.

CONCLUSIONS

1. It is necessary to distinguish between the fully grown and calcified adult tooth and the tooth that is still in the process of growth and formation.

2. It is fairly well established that faulty calcium metabolism results in no changes in the adult tooth except for an increase in calcium in the dentin with age.

3. There is no evidence that dental caries is aggravated by metabolic changes during pregnancy. There is no evidence that dental caries is more prevalent in the pregnant than the non-pregnant at the same age level.

4. Diet, calcium therapy, vitamin deficiency and endocrine dysfunction have no effect on the expectant mother's teeth. To aid the fetus, such deficiency must be corrected early and continued until the permanent teeth are erupted.

5. The use of calcium and vitamins in large doses is unwarranted. In both the expectant mother and the fetus, a well-balanced diet will make unnecessary the addition to the diet of most or all of the vitamin concentrates and calcium compounds that are being exploited, to the financial detriment of the patient and the confusion of the physician.

BIBLIOGRAPHY

1. READ, T. T.: *J. State Med.*, 43:445, August 1935.

2. GLANCKOPE, BERNARD: *Pub. Health Nursing*, 26:485, September 1934.

3. ZISKIN, D. E., and HOTELLING, HAROLD: *J. D. Res.*, 16:507, December 1937.

4. MATHIEU, ALBERT: *Northwest Med.*, 32: 292, July 1933.

5. REED, C. B.: Mouth in Pregnancy. J.A.D.A., 20:1631, September 1933.

6. ROSEBURY, THEODORE: *Arch. Path.*, 15: 260, February 1933.

7. BRODSKY, R. H.: Factors in Etiology and Arrest of Dental Caries. J.A.D.A., 20:1440, August 1933.

8. TAYLOR, G. F., and DAY, C. D. M.: *Brit. M. J.*, 1:919, May 6, 1939.

9. MARSHALL, J. A.: *Physiol. Rev.*, 19:389, July 1939.

10. KUGELMASS, I. N.: *New York State J. Med.*, 37:1733, October 15, 1937.

11. MULL, J. W.; BILL, A. H., and KINNEY, F. M.: *Am. J. Obst. & Gynec.*, 27:679, May 1934.

12. REED, C. B.: *Am. J. Obst. & Gynec.*, 26:814, December 1933.

13. SCHUMANN, E. A.: Textbook of Obstetrics. Philadelphia: W. B. Saunders Co., 1936.

14. Year Book of Obstetrics and Gynecology. (Edited by J. B. DeLee and J. B. Greenhill.) Chicago: Year Book Publishers, 1939.

15. DAY, C. D. M.: Effect of Antiscorbutic Deficiency on Pregnant Organism and Dental Tissues. J.A.D.A., 20:1745, October 1933.

16. SCHOUR, ISAAC: *J.A.M.A.*, 110:870, March 19, 1938.

17. CANTAROW, ABRAHAM, and TRUMPER, MAX: Clinical Biochemistry. Ed. 2. Philadelphia: W. B. Saunders Co., 1939.

18. KRONFELD, RUDOLF: Histopathology of Teeth and Surrounding Structures. Philadelphia: Lea and Febiger, 1939.

19. DEAN, H. T.: Mottled Enamel Survey of Bauxite, Arkansas, Ten Years Change in Common Water Supply. U. S. Gov't Printing Office, Washington, D. C., 1939.

20. MCKAY, F. S.: Mottled Enamel: Result of Change of Water Supply at Bauxite, Arkansas: Ten Years Afterward. J.A.D.A., 26:900, June 1939.

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IS DENTISTRY MEETING ITS OBLIGATION AS A PUBLIC HEALTH SERVICE?

THE attention that has been attracted to dentistry because of the supposedly inordinate percentage of defective teeth found in the mouths of the draftees in the present defensive emergency as compared to other disqualifying physical defects of the body has apparently raised the question in the minds of some as to whether dentistry is properly serving its health mission to humanity.

Because the percentage of rejections for military service attributable to dental deficiency is apparently larger than during the first World War, it is assumed in some quarters that the dentist has not done his duty in coping with the ravages of dental diseases; the implication being that if the dentist had advanced in his methods of handling disease as have other health specialties, he would have been better equipped to deal with disease in his field and, therefore, would have been more successful in combating it, or at least he would have been able to hold his own in the combat.

Other things being equal, this mode of reasoning would be quite sound, but other things are not equal.

Let us make a brief analytic study of the facts upon which this implication of dereliction of duty is based.

It is true that the percentage of rejections because of dental deficiencies under the present Selective Service System is greater than that of the Civil War and of the World War, but a study of the requirements exacted with regard to the dental mechanism will readily account for the apparent disparity. The estimate of physical deterioration or improvement in the dental status of the population, as is the case in the general health status, is based upon the increase or decrease of the number of draft rejections for general military duty in each of the three main recruiting efforts in the past century.

The draft rejections in the Civil War were 37 per cent of those who were examined for enlistment; in the World War I, it was 31 per cent, and, in the present defensive emergency, it has been 38 per cent. It is, therefore, assumed that the general health status, as well as the dental health status, improved materially from 1864 to 1916, and that it has deteriorated materially from 1916 to 1941, as presumedly shown by the increase of draft rejections in 1941, about 7 per cent.

The age periods covered by the drafts: The Civil War draft was from 20 to 45 years; the World War I, 21 to 30; the present defensive draft, 21 to 35. A comparison of these age periods with the percentages of rejections may throw some light on the apparent discrepancies.

In each of these instances, rejections for dental deficiencies were larger than for any other diseases or conditions. The number of rejections for dental deficiencies of the five-year age periods for the Civil War were: ages 20 to 25, eight rejections; ages 25 to 30, twenty rejections; ages 30 to 35, thirty-nine rejections. The succeeding periods showed a steep increase, until the period from 40 to 45 showed eighty-seven men rejected per thousand—a very definite progressive increase in rejections according to the five-year age periods.

In the World War, the percentage of rejections was 31 per cent and the age limitations were from 21 to 30.

In the present defensive effort, the age limitations are 21 to 35, and the first physical examinations were from a drafted personnel in which emotional and sentimental impulses played no part. Yet, under the much more indiscriminate drafting of men and with a much higher age limit, the percentage of rejections has been thirty-eight as compared to thirty-seven in 1862 and thirty-one in 1916.

In the Civil War, as in every instance in which the United States has taken measures toward war, the teeth and mouth played an important

rôle in the enlistment requirements. In fact, the teeth have been a main factor in limitation of the number of supposedly physically fit youths of the land. The age conditions and the urgency of circumstances, however, have, in the last analysis, been the determining factors in limiting the number of volunteers, conscriptees or draftees.

It should not be forgotten that examination methods and methods of diagnosis of disease both of the mouth and of the body generally have been greatly improved.

In 1916, in 86 per cent of cases of venereal disease, the persons affected were accepted for general military service. In the present draft, venereal disease is regarded as a cause for rejection until there has been recovery without disqualifying sequelae. And it is quite possible that if the present emergency continues and the need for men increases, many of the rules will be relaxed; and, as standards are lowered, the percentages of outright and of partial rejections will decrease.

To those who are inclined to take the figures at their face value, the nature of the most prevalent of dental diseases—dental caries—is evidently not considered; namely, the fact that dental caries finds its most ravaging expression in its cumulative damaging effects. In other words, the virulency of caries increases materially with age by virtue of its progressive nature. So true is this that the whole problem of prevention of caries is largely contingent on the age period at which the person comes under the ministration of dental preventive measures. And it has become a truism in dentistry that preventive measures are appreciably effective in mouths that come under the dentist's care as early as the second or third year of life. Of course, these early preventive measures are beyond application to the present-day recruit, but the results of early neglect are already indelibly imprinted in the mouths of these recruits.

Caries is not contagious in the strict meaning of the word, but decay can spread and its spread from one tooth to another is greatly facilitated by the presence of decay. So that in a group of persons in which the prevalence of decay might be considered as being equal, and ranging in age from 21 to 35 years, there would be much greater percentage of dental caries present than in a group of the same number ranging in ages from 20 to 30, and the dental literature is flush with statistics to prove that the later years show much more of the cumulative results of progressive caries.

Next, let us consider a psychologic influence of the cumulative results of dental disease over which the dentist has no control, or has not been

able to control, to any great extent, because of the fatalistic attitude of many people toward the teeth, engendered by the false conception of the inevitable fatality of caries to the tooth. This conception has precluded response to the educational efforts of dentistry. A lack of appreciation of mouth health and an indifferent and a fatalistic attitude toward dental disease on the part of the medical profession have had great influence on, and have been definitely reflected in, the attitude of the laity.

The reaction to Hunter's indictment of the dental profession in its failure to apply the principles of asepsis in the insertion of mechanical apparatus in the mouth was a deprecation of all effort to save teeth; and, because of that attitude, the teeth were practically abandoned to their fate—which was inevitable loss. Rather than encourage any effort to save teeth, the medical man did not hesitate to condemn any and all teeth that were in the least suspected of disease or were in any way related to systemic disturbance, and this idea was carried to such an extreme that almost all teeth were condemned after it had been shown that some systemic conditions could be accounted due to dental disease.

As a consequence, we have a definite recrudescence of the mechanical age of dentistry—the revival of prosthetic dentistry and partial denture construction. For the person who had neglected his teeth up to the thirtieth year, the only hope offered is reconciliation to the inevitable —gradual loss of almost all of his teeth and the final acceptance of a full artificial dental masticating apparatus.

Another definite factor in this question is the economic condition, which has been largely responsible for the universal neglect of the teeth during the period of depression. There is no question that one of the greatest calamities that has ever overtaken dentistry was the reaction of the public to the need for dental care during that period. We are now reaping the whirlwind of that neglect.

At the very time in their development when most of these youths who are now being rejected for military service should have received the best of dental service, large numbers of dentists were unemployed and crying for work. And we trust that the rehabilitation program that is now occupying the attention of the government will be used as a means of compensating in some measure for the laxness toward its health obligation that has been evident, so far as dentistry is concerned, virtually throughout its professional life.

The one fact that stands out conspicuously in the present emergency is that nearly 40 per cent of what should be the flower of our young

manhood is being rejected as unfit for military service; and the next important fact from the dental standpoint is that responsibility for the 20 per cent of these rejections, which have been attributed to dental deficiency, cannot be charged to any fault of the dental profession.

As for the lesson to be learned by the present health status of the young manhood of the nation, it is indeed unfortunate that it required a near war tragedy to bring these facts to light, but it will be even more unfortunate if the projected programs of public health do not provide a dental service that is based on the needs of the people as a whole.

ENROLLMENT IN DENTAL SCHOOLS

WE publish elsewhere in this issue the enrollment in the thirty-nine dental schools of the United States at the opening of the present academic year as compared with the enrollment in last year's *Dental Students' Register* as of October 15, 1940. The total present enrollment is 8,371 as compared with 7,720 last October, an increase of 651 students. It will be noted that thirty-four schools show an increase of a combined total of 717 and that five schools show a combined decrease of sixty-six, a net gain of 651. These figures may be subject to slight changes when the register for 1941 is ready for release.

The record is gratifying indeed. This enrollment is the largest in the last fifteen years. In common with many others, we have been concerned at the fact that the total number of graduates from the dental schools in 1941 was the smallest in the last twenty years and, with two exceptions, the smallest in forty-four years. We have also been concerned at the fact that, during the last decade, the ratio of dentists to population has decreased for the first time in the history of organized dentistry in this country. It has been estimated that it would take at least 2,250 graduates each year to meet the loss by death and retirement and the anticipated increase in population.

While we have been concerned, we have at no time been alarmed. We have always insisted that advancing educational requirements do not long affect enrollments in professional schools. American youth has a way of meeting any reasonable standards set for it. In 1935, by common consent, the dental schools of America set a minimum standard of two years of liberal arts' training, followed by a minimum of four academic years in a dental school. This action on the part of the schools was courageous and highly praiseworthy. The present enrollment gives

abundant proof that the downward trend, due in part to the new requirements, has been wholly overcome. The curve has been upward since 1936. The present enrollment of 2,492 first-year students, if repeated for the next three or four years, as we have every reason to believe it will be, will give us a supply of entrants to the profession which will restore the former ratio of dentists to population. No one can foresee how many dentists will be needed if society finds a way to care for the dental ills of all our people.

We shall publish later an analysis of the entrance credentials of the present first-year class, which the secretary of the Council on Dental Education promises us. We know, from last year's record, that in all probability considerably more than half of the new recruits will be shown to have had more than two years of liberal arts' training and that at least one-fourth of them already possess a bachelor's degree. Coupled with the announced plans of the Council on Dental Education for the establishment of a list of approved dental schools, the awakened interest on the part of well-trained youth in the study of dentistry should be encouraging to the entire profession.

BIBLIOGRAPHICAL

Development of Occlusion. A symposium of three papers presented at the University of Pennsylvania Bicentennial Conference. Philadelphia: University of Pennsylvania Press, 1941. **The Evolution of Dental Occlusion from Fish to Man.** By William K. Gregory, Ph.D., D.Sc.

IN this paper, Gregory sets forth evidence to refute Patten's contention that vertebrates were derived from paleozoic eurypterids, which would make them related to the crustaceans. He insists that jaws and the dental apparatus were not derived from locomotor appendages, as is the case of the crab and the lobster, but first appeared as a part of the skeletal system of the early chordates. Such early jaws, however, were parts of the protective exoskeleton and were probably concerned with respiration as well as with food sifting, since they were serially homologus with gill arches. The most anterior gill arch ultimately developed a cartilaginous core, which persists as Meckel's cartilage.

The inner jaw was covered with a series of sheathing bones, the most anterior of which ultimately bore teeth. This portion, at first relatively small, gradually enlarged until it formed the entire lower jaw, while two of the suppressed bones became the malleus and incus. In a similar manner, the tooth-bearing bones of the upper face increased in size until they came to form most of the subocular area.

The temporomandibular joint, a mammalian characteristic, came into existence only when the mandible reached and impinged upon the squamous bone. A slip of the temporalis, in which muscle it was embedded, remained as the meniscus. The jaw muscles were originally the controlling mechanism of the gill arches, employed to work them like a bellows; but, even before the appearance of the temporomandibular joint, these muscles divided into a masticating group and those controlling the hyoid.

The teeth are thought to have originated

as derivatives of the laminated exoskeleton as early as the ostracoderms. It was not until the shark stage was reached, however, that they assumed their conical forms. Then followed a period exhibiting great complexity in tooth form until the Jurassic period, when the tritubercular pattern appeared. This seems to be the ancestor of most modern tooth forms. The last part of Gregory's contribution deals with the transitions and transformations which have led to the teeth of modern animals.

Ontogenetic Development of Occlusion. By B. Holly Broadbent, D.D.S., F.A.C.D.

This portion of the symposium is a statement of the general findings derived from the labors of the Bolton Study of Development of the Face of the Growing Child, Department of Anatomy, Western Reserve University. As this reviewer has pointed out on previous occasions, the thoroughness and scope of these investigations stamp them as among the most illuminating ever to be conducted on the skeleton of the head.

In order to conform to the central theme of the symposium, Broadbent has largely restricted himself to a consideration of the development and eruption of the teeth and to those structures of the face most intimately concerned with their proper placement. However, he does not neglect to point out the importance of the normal growth of the face as an indispensable adjunct to normal occlusion. If the text were entirely eliminated from this presentation, the illustrations alone would tell the story of the ontogenetic development of man's dentition. There are twenty stages shown, beginning at 1 month and ending at 18 years. These are in the form of tracings of lateral head roentgenograms and all non-essential details have been eliminated. They show clearly the course of eruption of every tooth in both deciduous and permanent dentitions. In another section are shown the series of possible events when dentition age is in advance of skeletal age. The teeth, erupting into arches not quite ready to receive them, are thrown into somewhat grotesque arrangements, which Broadbent has aptly called "ugly duckling" stages. He shows how subsequent growth of the jaws permits the teeth to arrange themselves in their normal relations if the discrepancy has not been too great.

The final portion of the presentation deals with the nature of the growth changes exhibited by the normal child. The process is a steady and regular one, with all angular relations tending to remain constant and all anatomic points traveling in straight lines. Proportions do not change as each part increases in size.

Factors Influencing Occlusion. By Milo Hellman, D.D.S., Sc.D.

Hellman points out that the "basic patents" on normal occlusion were taken out by nature millions of years ago. The major principles which still exist in modern animals were exhibited by those mammal-like reptiles of the carboniferous era. In the most primitive of these forms, the teeth of the upper jaw overhung those of the lower and alternated with them in position. In the more advanced species, the crowns had developed one main cusp and two smaller accessory cusps. Even in these, the teeth of the two jaws alternated as in the modern. The next stage of development saw the main cone of the upper migrating toward the palate, while that of the lower moved buccally. Since these principal cones lay between the accessories in a mesiodistal direction, and since the upper and lower teeth alternated, the shifting of the great cusps brought them into the embrasure formed by two teeth of the opposite jaw. The connecting of the cusps by marginal ridges brought blades into play, and the shearing action of teeth was introduced. Up to this stage, teeth passed each other in closing in the same manner as shown today in the flesh eaters.

The next stage consisted of the development of a talon, or extra cusp. This formed in such a position that it was received in the fossa formed by the original triad of cusps, and henceforth the teeth rested upon each other when the jaws were closed. Long before this time, the dentition had been divided into incisors, canines, premolars and molars. As if now relieved of all restrictions that had been inherent in the "passing closure," Nature indulged in a wild orgy of cusp formation and these increased rapidly in number and complexity. Then a reverse trend set in and a simplification began, which appears still to be operating. As early as the Miocene period, the mammalian den-

tition was showing traits that stamp it as preanthropoid. The incisors were not unlike those of man, the premolars were strongly suggestive of bicuspids and the groove pattern of the molars were becoming more human.

By the time the anthropoid stage of evolution had been reached, the dentition had arrived at essentially the human level, with the exception of the canines. These teeth, long, powerful and conical, were received in diastemas of the opposing jaws. In the most primitive ape man, Plesianthropus Transvaalensis, the canines have changed their form, becoming shorter and wider mesiodistally, with a consequent elimination of the diastemas. The occlusion had become an even plane and the human stage had been reached.

Hellman closes his portion of the symposium by presenting evidence that modern dentitions reveal the same degree of variation that one would expect to find in any evolving organ. He compares primitive dentitions with modern ones by enumerating the number of contact points made by the teeth in occlusion. This indicates a diminution in number of such contacts, or further simplification. However, the ratio varies with different individuals, indicating that the endless experimentation of Nature is still going on.

ALLAN G. BRODIE.

Dental Caries. A symposium of three papers presented at the University of Pennsylvania Bicentennial Conference. Philadelphia: University of Pennsylvania Press, 1941. Price 50 cents. **The Diet in Relation to Dental Caries.** By Elmer V. McCollum, Ph.D., Sc.D.

THE great variation of susceptibility of different people to dental caries may be studied from its nutritional aspects. If the nutrition is optimal during the development of the teeth, the tooth structure will be safeguarded. Freedom from structural defects such as pits, fissures and hypoplastic enamel may be expected. A state of optimum nutrition may afford protection against dental caries. This protection may be due to some property of the mucous secretions or of the saliva which tends to suppress the growth and functions of microorganisms inimical to the health of teeth.

There seems to be evidence that the consumption of a diet in which the ratio of fatty acids to total carbohydrate is suitable for a diabetic affords protection against caries. This protection by excessive fat in the diet may be due to greasing the tooth surface. There seems to be evidence that the elimination of sugar from the diet, and the provision of carbohydrate in the form of starch, affords a less favorable oral medium for the development of acid-forming organisms.

The Bacteriology of Dental Caries. By Basil G. Bibby.

THERE are many reasons for believing that the presence of bacteria is fundamental to the initiation of dental caries: 1. Tooth destruction progresses from the outside of the teeth inward. 2. Caries occurs where foods accumulate. 3. An association has been shown to exist between the acidogenic properties (carbohydrate content) of diets and the development of caries. 4. As yet, acids are the only chemical or biologic agents known to have the power of destroying dental enamel and bringing about the decalcification of dentin. 5. Bacteria are the only agents existing in the mouth that are known to have the power to produce acid from foodstuffs.

In the author's opinion, any organism that can produce acid in the mouth may cause tooth decalcification, but their presence in large numbers, and the active chemical effect makes the rôle of streptococci of greater importance.

The reduction of dental caries by the action of fluorine, which is known both to interfere with acid production by microorganisms and to increase the acid resistance of enamel, may be considered as further evidence that caries is primarily the result of decalcification of dental tissues by acids formed through bacterial action in the mouth.

Epidemiology of Dental Caries. By Henry Klein and Carroll E. Palmer.

QUANTITATIVE measures of the tendency of populations to be attacked by a disease may be obtained from arithmetic ratios which give, per unit of the population, (a) the number of cases of the disease existing at any given moment of examination of the group, and (b) the number of new cases arising within particular intervals of time.

The discussion of the epidemiology of the caries tendency in this paper is based, in the main, on prevalence data. Certain procedures for measuring this tendency are described and the rationale justifying their use is set down. The dental statistics appearing in the literature are analyzed in terms of these basic definitions, and, from the resultant findings, an outline of the epidemiology of dental caries may be discerned.

The items reviewed indicate that the tendency to experience dental caries is considerably different for persons of various racial and family groups and for persons residing in different geographic areas of the United States. The mineral content of drinking water appears to be related to the caries tendency, and the ingestion of certain trace chemical elements may depress this tendency. On the other hand, the available findings appear to support the view that socio-economic conditions have little if any influence on vulnerability to carious attack.

K. A. EASLICK.

Standard Bodyparts Adjustment Guide. Insurance Statistical Service of North America. Chicago, Ill. Single copy $15, including ten years' revision service.

FROM the viewpoint of attracting dentists as purchasers, this book is poorly named because the title is not descriptive of its contents. It is largely a "Workmen's Compensation" book because it contains all of the workmen's compensation laws, a description of the injuries which are covered by the laws and a schedule of fees which are paid for medical and dental services. It presents other material along the same line.

The book is primarily designed for the use of physicians doing workmen's compensation work and insurance men working in this field. The most recent edition also includes information about dental injuries and sample fees paid by state compensation boards.

The book includes a series of excellent anatomic charts for reference to the various types of injuries and might be of considerable interest to those dentists who handle compensation work.

The original cost of the book is high, but the fact that this entitles the purchaser to a ten-year revision service makes the yearly cost quite reasonable. Any dentist who is interested in workmen's compensation and medical and dental care will find that this book contains a great deal of useful information. Dentists who are interested only in representative fee schedules can obtain information on this subject free of charge from the state compensation boards or the American Dental Association.

Pathology for Students and Practitioners of Dentistry. By William E. Ehrich, M.D., Associate in Pathology, School of Medicine, University of Pennsylvania. Philadelphia: Lea & Febiger. Cloth, 509 pages. 234 illustrations. Price $5.50.

MANY teachers of the fundamental sciences in dental schools have had difficulty in finding suitable textbooks for their courses because the average textbook used in medical schools is usually too detailed and too lengthy to make it ideal for dental classes; and a textbook written specifically for dental students is all too often superficial and incomplete.

Dr. Ehrich has attempted to solve this problem in the field of pathology by writing a book which illustrates general pathologic principles with material from the oral cavity. Most dental students and dental graduates should find the book interesting because the author discusses only pathologic conditions which directly concern dentists, mentioning diseases of organs remote from the mouth only when of importance to dentistry. In fact, the author has limited his field so definitely that many will criticize the book as incomplete.

The book is carefully organized and is tersely and clearly written. The illustrations are well chosen and of high caliber. An adequate bibliography, perhaps overcrowded with references to foreign journals, is included with each chapter.

Dental practitioners and students should find Dr. Ehrich's book an interesting introduction to oral pathology.

BOOKS RECEIVED

Odontologia e Medicina (Dentistry and Medicine).

By CIRNE LIMA, Professor of Pathology and Applied Therapeutics of the Faculty of Medicine of Porto Alegre. Published in two volumes, in the Portuguese language. Paper cover. Porto Alegre (Brazil). Livraria do Globo. 1939.

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BUREAU OF PUBLIC RELATIONS

THE FEDERAL GOVERNMENT AIDS THE CHILD*

By FRANK C. CADY,† D.D.S., M.P.H., Lexington, Ky.

IT is a pleasure to present to you the federal dental health activities relating to the welfare of the child, since we in the federal service can look back with some degree of satisfaction at the progress made over the last few years.

While much of this progress is not specifically measurable in terms of prevention of tooth loss, we do feel that there has been a rapid development of the machinery necessary to attack this tremendous problem. We also derive some satisfaction from the knowledge that we have in a measure fulfilled one of our major functions, which has been to increase interest and activity in this field among health organizations in the states and in local communities.

U. S. PUBLIC HEALTH SERVICE

A large part of the Federal activity in dental health is being carried on by the U. S. Public Health Service, which dates back to 1798. Created originally to provide medical care for sick and disabled seamen, its functions were extended by successive acts of Congress to include many other important duties, including research and measures for the protection of the public health. As far back as 1893, an act of Congress directed the Surgeon General of the Public Health Service to cooperate with and aid state and municipal health agencies in the prevention and control of disease.[1, 2] From this, it can be readily understood that joint responsibility in the field of health protection was not a new conception that arose with the passage of the Social Security Act in 1935. Not until the enactment of the Social Security Act, however, had there been sufficient recognition by the Federal Government of its share of responsibility in health matters.

It is evident that Congress, in considering this legislation, deemed it within what is known as the welfare class of the Federal Constitution (Par. 26, Section VII, Act 1).[2] In this respect, the public health title of the act differs from most of the existing law relating to the Public Health Service and is worthy of note. For the first time in the history of the public health movement in this country, Congress has made a declaration of permanent policy under which it assumes, in part, responsibility for protection of the individual and has made provision for participation of the Federal Government in the establishment and maintenance of health services for this purpose.

There is nothing untried or unproved in the procedures prescribed in the social security legislation for grants-in-aid to the states. The Public Health Service has been actively engaged in cooperation

Read before the Section on Dentistry for Children at the Seventy-Seventh Annual Midwinter Meeting of the Chicago Dental Society, February 18, 1941.

*Part of a symposium on "What Are Health Agencies Doing to Promote Dental Health for Children?"

†Senior Dental Surgeon, U. S. Public Health Service.

with the states in the organization and maintenance of state and local health service since 1918. This cooperation has been conducted through technical assistance to and the assignment of service officers, on request from states, to duty within the states.

It is most fortunate that the Public Health Service has by long experience been fortified in cooperative health work with the states, and, in like manner, the states themselves have acquired knowledge and experience in cooperative public health procedures. It is only through such actual experience that thorough preparation for meeting the problems involved in the administration of the public health provisions of the Social Security Act can be acquired. Both the Public Health Service and the states themselves have thus been prepared to proceed most effectively from the start, without having to resort to the unnecessary motion and expense of experimentation through trial and error.

Under the provisions of Title VI of the Social Security Act[1] as amended for the fiscal year 1941, authority is granted for:

1. An annual appropriation of $11,000,000 for the purpose of assisting states, counties and health districts and other political subdivisions of the states in the establishment and maintenance of adequate health services, including the training of personnel for state and local health work.

2. An annual appropriation of $2,000,000 to the Public Health Service for research activities of the service and for the expense of cooperation with the states in the administration of the federal funds granted for aid in the establishment and maintenance of state and local health services.

It is conceived to be the aim, among other purposes, of title VI of the act, to promote a comprehensive, nation-wide program of public health, financially and technically aided by the Federal Government, but supported, as far as possible, and administered by the states and local communities. To this end, the funds provided for allotment and payment to the states are available for:

(a) Strengthening state and territorial health departments, or bureaus or divisions of such departments.

(b) Through the state and territorial health departments, strengthening or aiding in the development of district, county and city health services.

(c) Training personnel employed, or to be employed, in state and local health departments.

THE CHILDREN'S BUREAU, LABOR DEPARTMENT

Another agency of the Federal Government concerned with the health and welfare is the Children's Bureau of the Department of Labor. As the name implies, its interests are largely centered in the health and welfare of children. Under the provisions of title V of the Social Security Act,[2] they receive an annual appropriation of $5,800,000 for the purpose of assisting the state and local health departments in the development of maternal and child health services. In addition, the Children's Bureau receives appropriations to aid crippled children and for other child welfare service.

From these grants-in-aid provided by titles V and VI of the Social Security Act, funds are allocated annually by the states for dental health services.

With the enactment of the Social Security Act of 1935 came recognition by the Federal Government of its share of responsibility in dental health matters. In addition to grants-in-aid to the states for the establishment of dental health services and the training of dental personnel, the Public Health Service organized a dental section in its states' relations division.[3, 4, 5] This section, which is directed by a dental officer with public health training and experience,

offers the following services to state and local health departments:

1. Consultation services on the organization and administration of dental health programs.

2. Funds and personnel to conduct graduate or refresher courses in dentistry for children for the benefit of practicing dentists.

3. A limited number of trained Public Health Service dental officers, on request from the states, assigned to duty within the states to organize new programs or projects.

Other dental health projects of the Public Health Service are to be found in the Division of Scientific Research, which is the National Institute of Health. Here, the Public Health Service conducts a number of studies directly related to the problems of dental disease. Problems being studied at present include the relationship of dental caries and endemic dental fluorosis, the effects of avitaminosis on dental structures and the dental aspects of industrial health hazards.[6] The division of public health methods of the institute has, during recent years, conducted studies and surveys on the epidemiology of dental caries in children. Only a brief report can be given of these various activities in this paper. Those interested in full reports of the conduct of the work to date can refer to the bibliography included with this paper.

The basic study of the epidemiology of endemic dental fluorosis has been almost completed. The major activity in this field at present is directed to a study of the relationship of endemic fluorosis to dental caries.[7, 8] At the present time, this study includes reports on 3,400 children, all of whom have a history of continuous exposure to various concentrations of fluorine in their water supply.

For some time, the National Institute of Health has been conducting studies on the experimental production of dental caries, nutritional studies on fluorides in food[9] and studies on the effects of fluorides on salivary amylase.[10] Additional data on these subjects will soon be published. The National Institute of Health has also made studies on the relationship of oral disease and dietary deficiencies.[11, 12] These experiments, which were made on monkeys, provide valuable information on mouth lesions directly associated with deficiencies in the diet of vitamins A, C, D and the B_2 complex. At the present time, studies are being made on the effects on monkeys of partial dietary deficiencies in vitamin C over a long period of time; also on the effects of calcium deficiency and a combination of calcium and vitamin C deficiencies on monkeys. The last three studies have not as yet been completed.

An entirely different type of research work is being carried on by the dental unit of the Division of Public Health Methods of the National Institute of Health. Here, trained dental officers study the prevalence of dental caries in the child population of the country as a whole, as well as the identification of the variations in prevalence of caries in specific localities.[13, 14, 15, 16] This group has also spent considerable time in the determination of basic definitions and methods by means of which the dental status of children can be accurately measured and ascertained.[17, 18, 19] Most of the field work of this dental unit has been done in Hagerstown, Md. Many of you are familiar with the reports of these basic studies. Altogether, ten different reports, based upon original data obtained in Hagerstown, have been published.

In recent years, a number of other groups and individuals of the Public Health Service have made significant contributions to the literature on dental health. Some of these have been taken from the National Health Survey conducted in 1935 by the U. S. Public Health Service with the aid of financial grants from the W. P. A. This survey

consisted of a house-to-house canvass of about 800,000 families, including 2,800,000 persons in eighty-three cities and twenty-three rural areas of nineteen states. Dental data obtained from this inventory have been culled by health workers and statisticians of the Public Health Service and published. One supplementary schedule of this survey having to do with the nature and extent of dental care and covering 20,000 families in Detroit, Mich., was reported by Britten.[20] This study included 66,000 persons of all ages over 3 and in all economic groups. The report shows that only 22 per cent of the total number visited a dentist for any reparative service during the study year and, of that small group, less than a third, or only about 4,800, or 7 per cent of the total number, were under 14 years of age. This is indicative of the very small number who go to a dentist at an age when an effective control service can be initiated.

Mountin and Flook also collected data from the National Health Inventory and evaluated some of the features of dental service as provided by more than 1,800 health agencies in ninety-four counties.[21] This survey revealed that these agencies were placing only about 15 per cent of the estimated fillings required for indigent school children in these communities. Other studies made by Collins and by Klein and Palmer,[22, 23] of the Public Health Service, definitely show the intimate relationship existing between economic status and volume of dental care received. In some instances, communities with a high economic index receive nearly five times the amount of service as other communities with a low economic index.

It is hardly necessary to mention to a dental audience the nation-wide dental survey made in 1933-1934 by the Public Health Service in cooperation with the American Dental Association of more than 1,400,000 school children in twenty-six states.[24] This study was the first to give us a good picture of the extent of dental defects existing in the child population. In all probability, the findings of this survey did more than any other one factor to arouse interest among non-dental groups in the dental problem.

These are the major surveys and studies made by the Public Health Service and having to do with the dental status of American children. Perhaps this is the major contribution that has been made by the Federal Government in the field of dental health for children, but it is not all. In addition to direct special services such as research, education and advisory assistance, the Federal Government has, since 1935, by authority of titles V and VI of the Social Security Act, provided many thousands of dollars annually to the states for the promotion of lay dental education and the provision of graduate professional education and dental service for children. This fiscal year, almost $500,000 has been allotted to the states for this purpose. While this is not a large amount, it has led to the appropriation of additional funds from state and local groups on a matching basis. It is not my purpose to infer that the Federal Government, or others, for that matter, have done more than scratch the surface of this vital problem.

It is encouraging, however, to look back only a decade and note the progress. Twenty years ago, the Federal Government was doing almost nothing in this field, and the states little more. Largely as a result of the stimulus of Federal money and effort, the number of dental health units in state health departments has increased in the last six years from twelve to forty. The number of personnel has more than tripled. This influence is also partly responsible for the expansion of these facilities in local communities. It is, I believe, safe to say that, largely as a result of this impetus,

there are few people in the country who have not received at least a modicum of dental health education.

BIBLIOGRAPHY

1. WALLER, C. E.: Social Security Act in Relation to Public Health. *J. Am. Pub. Health A.*, 25:1186-1194, November 1935.

2. Compilation of Social Security Law: Publication of Social Security Board, Federal Security Agency, Washington, D. C.

3. CADY, F. C.: Dental Organizations in State Departments of Health in U. S. Pub. Health Bull. No. 251. Government Printing Office, 1939.

4. *Idem:* Public Health Aspects of Dental Caries Problem. *J. Am. Pub. Health A.*, 25: 766-771, May 1939.

5. *Idem:* Selection and Training of Dental Health Personnel. *J. Am. Pub. Health A.*, 29: 328-333, April 1939.

6. Working Environment and Health of Workers in Bituminous Coal Mines, Non Ferrous Mines and Non Ferrous Smelters in Utah. Division of Industrial Hygiene, National Institute of Health and Utah State Board of Health.

7. DEAN, H. T., et al.: Domestic Water and Dental Caries. *Pub. Health Rep.*, 54:862-888, May 26, 1939.

9. DEAN, H. T.: Fluorine, Mottled Enamel and Dental Caries. *J. Pediat.*, 16:782-794, June 1940.

9. MCCLURE, F. J.: Fluorides in Food. National Institute Health Bull. No. 172. Government Printing Office, 1939.

10. *Idem:* Effects of Fluorides on Salivary Amylase. *Pub. Health Rep.*, 54:2165-2171, December 8, 1939.

11. TOPPING, N. H., and FRASER, H. F.: Mouth Lesions Associated with Dietary Deficiencies in Monkeys. *Pub. Health Rep.*, 54: 416-431, May 17, 1939.

12. TOMLISON, T. H.: Oral Pathology in Monkeys in Various Experimental Dietary Deficiencies. *Pub. Health Rep.*, 154:431-439, March 17, 1939.

13. KLEIN, HENRY; PALMER, C. E., and KNUTSON, J. W.: Studies on Dental Caries. I. Dental Status and Dental Needs of Elementary School Children. *Pub. Health Rep.*, 53:751-765, May 13, 1938.

14. KNUTSON, J. W., and KLEIN, HENRY: Studies on Dental Caries. IV. Tooth Mortality in Elementary School Children. *Pub. Health Rep.*, 53:1021-1032, June 24, 1938.

15. *Idem:* Studies on Dental Caries. VIII. Relative Incidence of Caries in Different Permanent Teeth. J.A.D.A., 25:1923-1934, December 1938.

16. KLEIN, HENRY, and PALMER, C. E.: Studies on Dental Caries. IX. Prevalence and Incidence of Dental Caries Experience, Dental Care and Carious Defects Requiring Treatment in High School Children. *Pub. Health Rep.*, 55:1258-1268, July 12, 1940.

17. KLEIN, HENRY; PALMER, C. E., and KRAMER, M.: Studies on Dental Caries. II. Use of the Normal Probability Curve for Expressing Age Distribution of Eruption of Permanent Teeth. *Growth*, 1:385-394, December 1937.

18. PALMER, C. E.; KLEIN, HENRY, and KRAMER, H.: Studies in Dental Caries. III. Method of Determining Post-Eruption Tooth Age. *Growth*, 2:149-158, August 1938.

19. KLEIN, HENRY, and PALMER, C. E.: Procedure for Recording and Statistical Processing of Dental Examination Findings. *J. D. Res.*, 19:243-256, June 1940.

20. BRITTEN, R. H.: Study of Dental Care in Detroit, Mich. *Pub. Health Rep.*, 53:446-459, March 25, 1938.

21. MOUNTIN, J. W., and FLOOK, E.: Dental Programs Sponsored by Health Agencies in Ninety-Four Counties. *Pub. Health Rep.*, 54:1625-1636, September 8, 1939.

22. COLLINS, S. D.: Frequency of Dental Service Among 9,000 Families. *Pub. Health Rep.*, 54:629-657, April 21, 1939.

23. KLEIN, HENRY, and PALMER, C. E.: Community Economic Status and Dental Problem of School Children. *Pub. Health Rep.*, 55:187-203, February 2, 1940.

24. MESSNER, C. T., et al.: Dental Survey of School Children Ages 6-14 Years. Public Health Bulletin No. 226, Government Printing Office, 1936.

HONOLULU INAUGURATES MOBILE DENTAL SERVICE

THE last word in trailers is now serving as a dentist's office for the school children of rural Oahu, Territory of Hawaii.

This new traveling dental unit was recently purchased by the City and County of Honolulu Health Department. It is reported to be the first of a fleet of such units contemplated for operation in the Territory, as a feature of the dental hygiene program now being carried on by the department of public instruction, various settlements and other agencies there.

The total cost of the mobile unit was approximately $9,000, including the latest type dental chairs; x-ray equipment; sterilizer; stainless steel sink; built-in cabinets; special lighting; inlaid linoleum; white enamel trim, and a comfortable leather davenport for waiting patients; making it the ultimate in convenience and sanitation.

New Honolulu dental trailer.

MOUTH HEALTH IN THE MAGAZINES

"YOUR Teeth and Your Children's" by Gladys Denny Schultz in the October issue of *Better Homes & Gardens* explodes some of the public's pet beliefs about tooth decay. In writing, Miss Schultz quotes freely from some of dentistry's better-known researchers on how to oust this enemy of family and dental health. "A nation," says Miss Schultz, "girding for defense—with physical fit-

ness all important—must look to its teeth." She then proceeds to explain why and what to look for.

Physical Culture also blossomed forth in October with "Faces Made Over by Dental Magic," by Wainwright Evans. Once people lived in dread of braces, bridges, false teeth and such. Today, according to Mr. Evans, modern science has discovered new ways to facial rejuvenation. This article attracts the attention with its clever arrangement of several before and after pictures.

Less spectacular, but perhaps even more helpful, was William R. Davis' article "What Can the Dental Health Worker Teach Regarding Nutrition and Diet?" in the July *American Journal of Public Health*. Because this journal is read by most health workers, from coast to coast, this article by Dr. Davis will do a world of good. In it, he debunks many dental dietary theories and offers some very sound advice on this controversial subject.

Thumb sucking is appraised from the mental hygiene and orthodontic points of view in the September issue of *Current Medical Digest* by Harriet Mitchell. This is a condensation of the original article that appeared in the *Canadian Medical Journal* earlier this year.

Which reminds us that the *Canadian Pharmaceutical Journal,* in September, carried a worth-while article by F. Norman Hughes on "Serving the Dental Profession." From a public relations standpoint, this is a most desirable presentation.

The American Baby carried A. Alan Greene's article on the care of the teeth in which he stresses the importance of keeping the foundation teeth in good condition.

The same subject with particular emphasis on correct mouth habits, during the preschool and infant years, was discussed by R. Reed Smith in the June issue of *You and Your Child.*

These are but a few of the recent magazine articles on dentistry that have been brought to our attention. They are mentioned here largely for your information, and also for the information of your patients. Their publication refutes the often repeated statement that magazines and newspaper editors are not interested in dental articles. They are interested if the articles are interesting.

BOOK REVIEW

The Long Winter Ends. By Newton G. Thomas, M.A., D.D.S., Chicago, Ill.

DENTISTS are usually interested when dentistry breaks into the literary field. They should be doubly interested when a dentist not only breaks, but also bursts into literary fame.

Newton G. Thomas, former Associate Editor of THE JOURNAL, did just that in September with his excellent book "The Long Winter Ends." Published less than sixty days ago by the Macmillan Company, it is already in its fourth printing and listed by several papers as a best seller.

Told with the utmost simplicity, "The Long Winter Ends" is a story of a young Cornish miner forced by the closing of his native mines to leave his young wife and unborn baby in a little Cornwall cottage, while he carved out a new home and a new life for them in America.

His experiences in the copper mines of Michigan, his triumphs over new world obstacles, even his laborious learning to write so that he could tell Pol, his young wife, about America, gave him a growing faith in his ability to create a future for himself and family in a country in which miners may become engineers, laborers, lawyers and green "Cousin Jacks" leaders of men.

COUNCIL ON DENTAL THERAPEUTICS

SOLUTION 58 (HANKE)—NOT ACCEPTABLE FOR A.D.R.

[Report of the Council on Dental Therapeutics of the American Dental Association, authorized for publication.]

THE Council's report on Solution 58 (Hanke) was submitted to Medadent Products Company in order that the firm might have an opportunity to reply in detail to the various points brought out in the Council's consideration of the product. After some delay, the firm presented reports of toxicity studies on rats conducted by Milton Theo. Hanke. The firm has not presented any further evidence on the efficacy of Solution 58 (Hanke) in the treatment of dental diseases, nor has it offered any comments valid in the light of the Council's rules regarding the name Solution 58 (Hanke). The Council referee and two consultants have carefully examined the toxicity data on Solution 58 (Hanke). The examiners are agreed that the evidence presented, which unfortunately was obtained after the product was put on the market, goes far toward establishing the safety of the preparation. They are not agreed, however, that the evidence is conclusive, particularly in that the experimental animals were on a high protein diet during administration of the drug and also no results of microscopic examination of kidney tissues were presented. In addition, the referee and his consultants believe that the transfer of data from experiments on rats to situations in which human beings are involved is not an entirely valid procedure.

The Council concurred in the referee's recommendations, and voted to present its original report, together with the foregoing introductory comments. The Council's report follows:

After publication of "Studies on the Local Factors in Dental Caries. I. Destruction of Plaques and Retardation of Bacterial Growth in the Oral Cavity," the Council received requests for information on Solution 58 (Hanke) and on sodium parahydroxymercuribenzoate. More recently, the Medadent Company of Chicago, for which Milton Theo. Hanke apparently acts as consultant, has placed a preparation called Solution 58 (Hanke) on the market. As far as the Council is aware, the sale of the product prepared by the Medadent Company is restricted to the State of Illinois. Apparently, a permit for interstate distribution of Solution 58 (Hanke) has not been granted by the Food and Drug Administration. It has come to the Council's attention, however, that other firms are marketing sodium parahydroxymercuribenzoate in packages labeled "Not for Drug Use."

In the light of these facts, the Council has requested a referee to examine the published information on sodium parahydroxymercuribenzoate and on Solution 58 (Hanke), with a view to presenting an informative report to the dental profession.

The referee has studied the available information on sodium parahydroxymercuribenzoate and on Solution 58 (Hanke). He has also enlisted the aid of several consultants whose comments have been of value in the preparation of this report. The referee and the Council consultants are agreed that the consideration of Solution 58 (Hanke) should involve a careful study of the safety and the effectiveness of the product under conditions of use.

A review of the available information on Solution 58 (Hanke) and on sodium parahydroxymercuribenzoate failed to

furnish adequate evidence upon which a claim for safety of Solution 58 (Hanke) might be based. Apparently, data on acute and chronic toxicity of the mercurial are not available. Nor does there appear to be any evidence on the rate and extent of absorption of the mercurial from the oral mucosa. These are not matters which can be dismissed lightly regardless of whether the mercurial is effective against acidogenic oral organisms. The referee is aware of Hanke's statement that "one-third ounce of Solution 58 contains only one-fiftieth grain of mercury, the major part of which is probably expectorated." Statements of this sort, which are really arithmetical assumptions, are of no value in attempting to answer the important questions relating to chronic mercurial poisoning. The mere fact that some people have used Solution 58 for a period of a few years without presenting any symptoms of mercury poisoning is only presumptive evidence and is therefore unacceptable. Statements of this kind are of no value whatever in formulating answers to such important questions as these:

What are the local and systemic effects of Solution 58 on healthy persons who may use the product over a period of twenty or thirty years or more?

What are the local and systemic effects of the product on persons whose kidneys are already damaged by disease to the extent that the kidney reserve is reduced nearly to the minimum physiologic requirements?

What effect may Solution 58 have on children and on incompetents who may swallow the product?

Cases of acute and chronic mercury poisoning have been described in the medical literature at various times. Many of these cases of poisoning have come about from excessive therapeutic use of mercurials. In other cases, mercurials have been swallowed with suicidal intent. In still others, the poisoning has been occupational in character. In a large number of instances, the poisoning has been due to metallic mercury or inorganic mercurials. The toxicity of the organic mercurials of which sodium parahydroxymercuribenzoate is an example is relatively low. But whatever type of mercury compound is used, it may give rise to mercury intoxication under favorable conditions. Moreover, it is not possible to place complete reliance upon oral symptoms for detection of chronic mercury poisoning. The symptoms may include loss of appetite, nausea and gastrointestinal conditions, pain in the bones and joints, anemia, loss of weight and general cachexia.

Metallic poisoning, including mercurial poisoning, has been discussed at length in a number of textbooks on pharmacology and in books on toxicology. The following excerpt from Cushny's "Pharmacology and Therapeutics," revised by Edmunds and Gunn (Lea & Febiger, Philadelphia, 1928), page 652, is pertinent to this discussion:

> Chronic Poisoning.—A much more frequently observed form of poisoning is that induced by the prolonged medicinal use of mercury. It may arise from any of the preparations and from any form of application, although some methods of administration are credited with being less liable to induce it than others. Thus inunction with mercurial ointment and the use of calomel internally are both more liable to cause the severer forms of stomatitis than is corrosive sublimate. A single hypodermic injection of an insoluble preparation may induce it in susceptible persons, because the mercury is only slowly absorbed, and passes into the tissues as gradually as if it were given by the mouth regularly for several days. This chronic poisoning, or *Mercurialism*, is due, not to the local action, but to the effects of the drug after absorption. It may follow the abuse of mercury in any case, but some individuals exhibit a special susceptibility from some unknown cause.

An examination of the statements in Hanke's paper relative to the effective-

ness of Solution 58 (Hanke) failed to reveal adequate evidence upon which any claims might be based. In fact, the purported clinical evidence was contained in the bald statement: "Dental caries has been definitely arrested or ameliorated in fifteen patients (in an unclassified group of 95) who have used the solution consistently as indicated above and who had always heretofore been highly susceptible to this disease." The statement is worthless as evidence. It meets none of the requirements for clinical evidence.

Hanke's data on the effect of the mercurial on acid production in saliva are interesting, but require confirmation. Several tests for susceptibility to caries, based upon chemical and bacteriologic technics, have been described in the literature and most of these antedate Hanke's findings. They have been discussed concisely in the book "Dental Caries" and do not require extended discussion here. Results obtained by these tests, although helpful, are not sufficient in themselves for an evaluation of agents used for the prevention or mitigation of dental caries. For this purpose, adequate clinical data obtained under controlled conditions are required. Other types of evidence may be valuable and research of this type should be encouraged.

It is claimed that the use of Solution 58 once or twice a day prevents formation of plaques on the teeth and that gingivitis usually disappears in a few days "except in those regions contiguous to calculus and even here the tissue tends to shrink to nearly normal." Also, it is claimed that "Solution 58 has also been used in a few cases of acute ulcerative gingivitis with very encouraging results." Statements of this sort read like testimonials and, in the absence of supporting data, must be so considered.

It is pertinent to inquire whether Solution 58 minus the mercurial might not have yielded results upon which the same type of statement could be made. At any rate, in view of the probable alkalinity of the solution and because of similar cleansing claims for lime water, it would appear logical to use such a solution as a control in experiments with Solution 58. In fact, why was not a suitable control used in the experiments on dental caries?

The referee does not believe that it is necessary to discuss Hanke's statements on the dental plaque beyond expressing the opinion that they do not constitute a significant addition to the dental literature. The same may be said of his qualitative experiments on the effect of acids on dental enamel and on the hydrogen ion concentration of plaques. More adequate studies on these topics have been presented elsewhere.

The referee cannot agree that the designation Solution 58 (Hanke) is appropriate or desirable. The name fails to comply with Rule 8 in that it does not indicate the chemical composition of the solution. Also, it is difficult to see how such a name would be in the interest of public welfare. Apparently, the only advantage in the name is a commercial one.

It is not the referee's intention to imply that no oral antiseptics of the mouth wash variety will ever be of value in the prevention or mitigation of dental caries. Indeed, some of the numerous findings in the literature could lend encouragement to the thought that a remedy of this sort may eventually be developed. Of course, there are other findings in the literature of a contrary nature. The only sound basis upon which products of this type should be offered the dental profession and the public is that of adequate scientific evidence.

In summary, the referee finds that the evidence regarding the safety and effectiveness of Solution 58 (Hanke) is inadequate and that the name of the product is in conflict with the provisions of Rule 8. He therefore recommends that

the product be held not acceptable for A.D.R. and that this report be published for the information of the dental profession.

A copy of the rules that govern the Council in the consideration of articles will be sent on request.

HAROLD L. HANSEN, *Secretary*.

ACCEPTED DENTAL REMEDIES

DENTIFRICES*

The Council on Dental Therapeutics of the American Dental Association announces the inclusion of the following articles in the list of Accepted Dental Remedies.

Sheffield Tooth Powder: Composition: Each hundred grams is stated to contain precipitated calcium carbonate (Snow Top Light), 90.40 gm.; Conti's castile soap powder, 7.30 gm.; saccharin soluble, U.S.P., 0.15 gm.; methyl salicylate, 2.05 gm.; oil of cloves, U.S.P., 0.05 gm.; menthol, 0.05 gm.

ABRASIVENESS: 0.00025 mg. loss on silver block under weight of 4,536 gm. in 400 revolutions.

Manufactured by the Sheffield Company, New London, Conn.

CALCIUM COMPOUNDS†

Non-Fer-Al Brand Calcium Carbonate: Non-Fer-Al Brand Calcium Carbonate complies with U.S.P. XI. It is used in the manufacture of dentifrices.

Manufactured by Diamond Alkali Company, Pure Calcium Products Division, Painesville, Ohio.

CHEMICAL ANALYSIS: 98.15 per cent calcium carbonate.

PHYSICAL PROPERTIES:

Specific gravity: 2.65.

Particle size:

Diameter in microns

Less than 5 microns	5-10 microns	10-20 microns	20-30 microns
44%	30%	20%	6%

Maximum size particle observed:

25 microns—single particle

50 microns—aggregate

Most of the particles over 5 microns in diameter appear to be aggregates not dispersed by the liquid used in the test.

ABRASIVENESS: The abrasiveness of Non-Fer-Al Brand Calcium Carbonate was determined by rotating a polished surface of antimony in a slurry of chalk and water under a weight of 63 gm. for 6,240 revolutions on a test machine (details filed with Council). The area of the metal specimens exposed was 53 square millimeters and the Bierbaum hardness was 236 units for antimony. The weight loss was 5.9 mg.

Admission of an article to the list of Accepted Dental Remedies does not imply a recommendation. Information regarding the rules and work of the Council will be sent on request.

HAROLD L. HANSEN, *Secretary*.

*A.D.R. Ed. 7, p. 102.
†A.D.R. Ed. 7, p. 87.

NATIONAL HEALTH PROGRAM COMMITTEE

PLANS FOR REHABILITATION OF REJECTED DRAFTEES

IN his press conference, October 10, President Roosevelt criticized the nation for permitting conditions which have left 50 per cent of its youth unfit mentally or physically for Army service and inaugurated a program to "salvage" 200,000 of the 1,000,000 youths who have been rejected.

Under the salvage program, the federal government will pay medical costs for treatment by local physicians of approximately 200,000 registrants whom local draft boards certify as susceptible of rehabilitation for Army service. The Army expects to accept virtually all of these 200,000 after they undergo treatment by family physicians or dentists at federal expense, Mr. Roosevelt said.

Describing the salvage program as only the initial objective, Mr. Roosevelt said that existence of conditions which permit so high a ratio of rejections is an indictment of America. He said he would launch a long-range program calling for cooperation of states, counties, cities, townships and individuals to remedy the conditions which are the underlying cause of the bad situation. He released statistics collected by Selective Service Headquarters showing that approximately 1,000,000 men have been disqualified for Army service because of physical, mental or educational conditions. This represents approximately 50 per cent of the total number of men examined. Mr. Roosevelt made it clear that he feared this ratio among men of draft age represented a fair index of the health conditions of all Americans.

The President read from a report presented by Brig. Gen. Lewis B. Hershey, Selective Service director, which declared that of the 1,000,000 rejected about 200,000 can be completely rehabilitated and made available for general service in our armed forces. The remainder can be rehabilitated to perform only limited service or, because of mental, nervous, cardiovascular and pulmonary diseases and musculoskeletal defects, are incapable of rehabilitation or even limited service and are therefore not being considered under the present rehabilitation program for Selective Service registrants, Hershey's report said. It continues:

"Our initial objective in this rehabilitation program will be the 200,000 registrants who can be completely rehabilitated and made available for general military service in the armed forces at a small cost and in a reasonably short time.

"Certain types of venereal diseases, operable hernias, deficiencies in vision and teeth, and other minor defects will be corrected in cases where the Army determines that the registrant will then be acceptable for general military service.

"The registrant will have the privilege of having the services performed by his family physician or dentist in his own community.

"The cost of this rehabilitation program will be borne by the federal government as a necessary part of our national defense program, and additional funds will be made available to the Selective Service System for this purpose."

Mr. Roosevelt was asked by what authority registrants could be compelled to undergo medical or dental treatment to prepare themselves physically for military service. He replied that when a registrant appears before an examining board, he is under the jurisdiction of that board and can be directed to undergo

such treatment. Conjecturing that a majority of such persons would be willing and eager to receive free treatment, Mr. Roosevelt said that in cases of recalcitrancy, the registrant could be inducted into the Army, placed under Army orders and commanded to undergo treatment. The President disclosed that the Army also had presented an alternative program, which he rejected, calling for induction of all rehabilitable cases and their treatment in Army medical centers.

When costs of housing, food, clothing, Army pay and medical care in this program were considered, Mr. Roosevelt said they would involve expenditure of approximately $500,000,000. The salvage program by local physicians and dentists which he has approved will cost far less than that, he said.

The President disclosed that plans already are well advanced for the salvage program. In cases of heart and musculoskeletal diseases, as well as mental and nervous cases, persons considered by local boards as being susceptible of rehabilitation will be placed in a special Selective Service category. Remaining under orders of their selection boards, these men will be visited by traveling boards or "teams" of prominent specialists, who will examine them and recommend curable cases for immediate treatment at government cost.

The Selective Service report itemized as causes for rejection:

Dental defects, 188,000 cases, 20.9 per cent.

Defective eyes, 123,000 cases, 13.7 per cent.

Cardiovascular diseases, 96,000 cases, 10.6 per cent.

Musculoskeletal defects, 61,000 cases, 6.8 per cent.

Venereal diseases, 57,000 cases, 6.3 per cent.

Mental and nervous diseases, 57,000 cases, 6.3 per cent.

Hernia, 56,000 cases, 6.2 per cent.

Defects of ears, 41,000 cases, 4.6 per cent.

Defects of feet, 36,000 cases, 4.0 per cent.

Defective lungs, including tuberculosis, 26,000 cases, 2.9 per cent.

Miscellaneous, 159,000 cases, 17.7 per cent.

The President said that in the cases of dental defects, hernias, eye trouble and even cardiac and muculoskeletal defects, the ratio of cures is expected to be relatively high. Venereal cases are especially susceptible of salvage, he said, and venereal infected registrants will be kept under the orders of selection boards and instructed to get themselves cured quickly and report back for service. The salvage program, Mr. Roosevelt admitted, leaves for future consideration the larger question of why half of American youths are physically and mentally defective and why 100,000 had to be rejected because of a lack of a fourth grade education.

Col. Leonard G. Rowntree, chief of the Selective Service Medical Division, after a conference with the President, said that the preliminary figures point to the inescapable conclusion that there is an urgent need for a national campaign for improvement of the general health.

[It is interesting to note that the Rehabilitation Program offered herewith as a governmental project conforms quite closely in principle and content to that which was presented by the National Health Program Committee to the Trustees of the American Dental Association at a special meeting in August of this year and published in THE JOURNAL for September.—ED.]

RESEARCH COMMISSION

LIST OF CERTIFIED DENTAL IMPRESSION COMPOUNDS

(A. D. A. Specification No. 3—First Revision 1939)

THE impression compounds listed below have been certified to the Research Commission by their respective manufacturers, tested at the National Bureau of Standards and found to comply with the requirements of American Dental Association Specification No. 3, first revision.

Compound	Type	Manufacturer or Distributor
Kerr No. 2 Soft Green	I	Kerr Dental Manufacturing Co.
Kerr Perfection	I	Kerr Dental Manufacturing Co.
Mizzy Low Heat	I	Mizzy, Inc.
Smith Certified	I	Lee S. Smith and Son, Mfg. Co.
S. S. White Tray, Black	II	The S. S. White Dental Mfg. Co.

REPORT OF THE RESEARCH COMMISSION

(1940-1941)

DURING the past year, the Research Commission established a Fellowship in Dental Diseases at the National Institute of Health in Washington, D. C. The formal presentation of the initial grant of $5,000 was made to the Surgeon General of the United States Public Health Service, Thomas Parran, January 14, 1941. The entire membership of the Executive Board of the Research Commission was present in Dr. Parran's office on this occasion and they had a very satisfactory discussion of the objects and functions of the fellowship. There was considerable delay in the completion of the signing of the contract for the fellowship owing to the fact that it had to be circularized to all members of the National Advisory Health Council, which is connected with the supervision of the work at the National Institute of Health.

It was not until May 31 that actual work on the fellowship could be started. A special room, fully equipped, has been set aside in the Division of Infectious Diseases for the Fellow in Dental Diseases. He will work under the direct supervision of H. Trendley Dean, a member of the Research Commission and chief of dental research at the National Institute of Health.

During the year, the second edition of "Dental Caries" was completed, and it is planned to publish this book before September 1. The success and general acceptance of the first edition assures us of the usefulness of this new volume. It is expected that this book will be of special benefit and assistance to the Research Fellow in Dental Diseases at the National Institute of Health. Elsewhere in this report will be found a detailed statement of the work of the Advisory Committee on Research in Dental Caries for the year.

REPORT OF THE AMERICAN DENTAL RESEARCH FELLOWSHIP AT THE NATIONAL BUREAU OF STANDARDS

THE Research Commission is again pleased to report the activities of its fellowship at the National Bureau of Standards. This fellowship, during the last fiscal year, had the following active representatives: George C. Paffenbarger, dentist; William T. Sweeney, physicist; John R. Beall, mechanical engineer, and H. J. Caul, chemical engineer.

From August 1, the fellowship will be without the services of Mr. Sweeney, who has accepted a position with the Vernon-Benshoff Company of Pittsburgh, Pa., where he will be in charge of research and testing. During his nineteen years of service, first as a member of the staff of the National Bureau of Standards, and then as Research Associate for the American Dental Association, Mr. Sweeney has been the author or coauthor of approximately thirty-five papers. To find a man to take his place in the Research Commission program will not be an easy task. The commission hopes to add an additional fellow at some time during the next fiscal year. There is a possibility that the fellowship will be further depleted of trained men, since Dr. Paffenbarger may be called to active duty in the Naval Dental Corps.

We are pleased to submit the following outline of the active work of the fellowship. This report, by the Senior Research Associate, includes the work of all of the research associates and that of the Bureau staff.

TECHNICAL REPORT OF THE AMERICAN DENTAL ASSOCIATION RESEARCH FELLOWSHIP AT THE NATIONAL BUREAU OF STANDARDS (1940-1941)

The cooperative research on dental materials for the fiscal year of 1940-1941 includes:

a. The testing of: 1. Certified materials. 2. Denture base materials. 3. Base metal alloys for prosthesis.

b. Miscellaneous activities.

CERTIFIED MATERIALS

The results of the testing of certified materials during the year are given in the accompanying table.

RESULTS OF TESTS OF MATERIAL FOR 1940-1941

Material	Number of Brands Tested	Number Complying with Specification	Number Failing to Comply with Specification
Amalgam	4	4	0
Casting gold alloys (inlay)	7	6	1
Casting investment (inlay)	8	8	0
Mercury	1	1	0
Wrought gold wire	4	1	3
Zinc phosphate	1	0	1
Hydrocolloidal impression material	8	5	3
Acrylic resins	2	1	1
Total	35	26	9

DENTURE BASE MATERIALS

Investigation of the denture base materials was continued and a report prepared for publication. The report includes data on the physical properties of thirty denture base materials, including methyl methacrylate resin, acrylic-styrene resin, acrylic-vinyl resin, cellulose and hard rubber. Several of the methyl methacrylate resins were tested in both the plastic cake and the powder-liquid forms. Shelf life, packing test, curing temperature, index of refraction and boiling range of the uncured materials were investigated. Water sorption, solubility, color stability, strength and stiffness in the transverse test, surface hard-

ness and softening temperatures of the cured materials were determined. The methyl methacrylate resin and the acrylic-vinyl copolymers were found to have the most satisfactory properties.

The data in this report were used as the basis for the formulation of the American Dental Specification No. 12 for acrylic resin for denture bases. This specification includes tests for translucency, satisfactory working qualities, porosity, water sorption, solubility and color stability. The specification was made effective in February and the testing of certified materials for compliance with it is in progress.

BASE METAL ALLOYS

Detailed chemical analyses of vitallium, ticonium, lunorium and niranium were completed. All the alloys contained chromium and cobalt in appreciable quantities, modified with various metallic elements such as nickel, beryllium, tungsten and molybdenum. Tests on the mechanical properties of these alloys are in progress.

MISCELLANEOUS ACTIVITIES

1. Reports on the program of the Commission and the work of the Fellowship were presented before ten dental organizations.

2. Visitors from China and Costa Rica were among the 113 who called at the laboratory during the year.

3. Publications for the year follow:

Research Commission: List of Certified Dental Materials. J.A.D.A., 27:791, May 1940.

Research Commission: Acrylic Resins. J.A.D.A., 27:960, June 1940.

BEALL, J. R.: Revision of American Dental Association Specification No. 4 for Inlay Casting Wax. J.A.D.A., 27:1140, July 1940.

SWEENEY, W. T., and CAUL, H. J.: Denture Rubber: Composition, Properties and Specification. J.A.D.A., 27:1446, September 1940.

Research Commission: List of Certified Inlay Casting Investments. J.A.D.A., 27:1518, September 1940.

PAFFENBARGER, G. C., and CAUL H. J.: Dental Cements. Proceedings of the Dental Centenary Celebrations. Baltimore: Waverly Press, pp. 232-237.

PAFFENBARGER, G. C.: Silicate Cement: An Investigation by Group of Practicing Dentists Under the Direction of A. D. A. Research Fellowship at National Bureau of Standards. J.A.D.A., 27:1611, October 1940.

Idem: Dental Research at the National Bureau of Standards. *Science*, 92:527, 1940.

SWEENEY, W. T., and PAFFENBARGER, G. C.: Tentative American Dental Association Specification No. 12 for Denture Base Material, Acrylic Resin or Mixtures of Acrylics and Other Resins. J.A.D.A., 28:325, February 1941.

American Dental Association Research Fellowship at National Bureau of Standards. J.A.D.A., 28:658, April 1941.

4. At the request of the Research Commission, the National Bureau of Standards has assembled and unified pertinent data from the more than 100 reports that have been issued on dental materials and dental technics. This will be published in book form by the Government Printing Office under the authorship of Wilmer Souder and George C. Paffenbarger. It will be available from the Superintendent of Documents some time during 1942.

GEORGE C. PAFFENBARGER,
Research Associate.

REPORT OF THE ADVISORY COMMITTEE ON RESEARCH IN DENTAL CARIES

PURSUANT to the proposals in my reports since the publication of the first edition of "Dental Caries" in 1939, and to the Commission's related authorizations, a second edition has been prepared in accord with the form and procedures for the first edition. Collection of the material for this edition has been in progress continuously since March 1940. Copies will be ready for distribution in

September. We have again received excellent cooperation from the Lancaster Press, which executed expertly the difficult work of typographic revision of the standing type of the first edition, and the artistic and economic combination of that revised material with the new matter for the second edition. The second edition, like the first, is a notable example of the art of book-making at its best.

The general outcome of our effort may be seen from this comparison: The first edition, issued two years ago, presented summaries of findings and conclusions on the causes and control of caries by 195 authors or groups of authors in twenty-five countries. It contained 189 pages—147 presenting the summaries (average, 0.75 page). The second edition will present not only revisions of many of the summaries in the first edition, the rest remaining unchanged, but also summaries by forty-two additional authors or groups of authors in twelve countries; a total of 237 in twenty-six countries. It will contain 277 pages, 209 presenting the summaries (average, 0.88 page).

In the second edition, each summary by a new author is marked with two asterisks (**). Each summary (in the first edition) that was revised by its author is marked with one asterisk (*). The rest of the summaries in the first edition, which were reprinted unchanged, are unmarked. A "supplementary analysis" (eighteen pages) of the new matter in the second edition follows the reprinted "general analysis" (sixteen pages) in the first edition. These arrangements facilitate examination by the reader of the new material separately, and also of both the old and the new together. In this edition, as in the first, there is no attempt in the analyses to evaluate the findings and conclusions in the book or to suggest which views may be regarded as most significant. Presumably, those functions will be performed publicly by reviewers and special students of caries and privately by critical readers. In this relation, we aimed only to compile the data in a readily accessible form, with matter-of-fact analyses of the contents, for constructive use by all who will promote public discussion or advance further clinical inquiry and research. The preface includes this comment:

"No minimum requirements for admission to this composite authorship were indicated or applied. The summaries of all who responded to our public and personal invitations were accepted into this compilation." It has been suggested by some that this liberal policy encouraged publication of a few "fantastic" views by several "rocking-chair scientists," and that only those who achieved accredited expertness in dental research should have been permitted to participate. We believe, however, that our presentation of this open opportunity, to all concerned, represents the spirit of true research and that our procedure has been more favorable for the advancement of research in dental caries —the main purpose of this book—than any restrictive or selective alternative might have been. Justice Oliver Wendell Holmes, in one of his most persuasive opinions, said, with universal acclaim, "The best test of truth is the power of the thought (finding) to get itself accepted in the competition of the market." "Error of opinion may be tolerated where reason is left free to combat it." (Jefferson, 1801.) There need be no fear that any views, unsupported by convincing evidence, will be seriously misleading. Researches in the future as in the past will nullify misjudgments. Thirty years ago salivary sulfocyanate was regarded by many as *the* etiological factor in caries. Twenty-five years ago a "salivary coefficient" (alkalinity) "provided the clue." In this volume no one mentions either claim—both unfounded and forgotten. In every *long* race between Truth and Error, Truth *always* wins.

It is not generally understood that "Dental Caries" created a new form of scientific literature. Its contents are not subject to bias or accidental distortion by an abstractor or reviewer, nor to an

editor's prejudgment, propaganda or censorship. It is not a collection of reviews or abstracts of particular publications. It presents brief and concise original summaries of the findings and conclusions of all individual observers or groups of workers who, on a world-wide "open invitation," crystallized their views in their own way as the authors for associated publication in this book —with appended references indicating where related details in their prior publications can be found. Arranged in the alphabetic order of the surnames of the authors, these readily accessible summaries are a series in one volume of many independent personal publications on the causes and control of caries. The book answers this question: Where can one find a summary of an author's findings and conclusions, which, as of a recent date and stated by him, are regarded by him as the outstanding, enduring, basic results of his experience in the study of caries, and which represent what the reader would now be justified in deducing from all of that author's related previous publications on this subject? To illustrate: Bunting's summary of the outcome of his many published studies of caries, as he now evaluates the findings, occupies only three pages.

"Dental Caries" presents the realities in the accumulated knowledge and contradictions on the causes and control of caries; affords opportunity for close and convenient study of the situation by every practitioner who wishes to ascertain the strength or weakness of current theories or procedures, and indicates general and specific avenues for further research in many relationships. The book has been compiled to promote critical study, understanding, clinical observation and research in this field. It promises to achieve these purposes.

The last two paragraphs of the "Supplementary Analysis," which discuss the general situation shown by "Dental Caries," present the following comment on "things as they are":

The concluding paragraph in the first edition of this book contained an allusion to the urgent need, in further research, for more and brighter light on the path to prevention of caries. The situation, as shown in the present edition, emphasizes this need for additional inquiry. Are ways to prevent caries included, unrecognized or unaccredited, in the recorded disagreements? Are there any missing links in the accumulated findings and conclusions? Are means for prevention dependent upon conditions that have not yet been noted or anticipated? Is caries, like headache, a result of any of a variety of unrelated causes? Each of these possibilities, and others, should be a stimulus for intensive researches, sustained economically by public and philanthropic support. Meanwhile it will be necessary to reiterate, frequently and widely, that agreement as to ways and means to "prevent caries, through establishment of conditions that would make its onset in any person impossible" has not yet been placed upon a scientific foundation.

This book presents a record of notable endeavors and achievements, by many earnest and able men and women, in dental research. As in other fields of research, remaining obstacles must be removed before complete success can be achieved. Thus, in medicine, the causes and control of many degenerative, metabolic, infectious and other types of widely prevalent disorders continue, despite active and prolonged research, to elude understanding. Ailments in regions near the teeth—eye, ear, nose, throat and scalp, for example—afford instructive comparisons. Glasses are worn more commonly than ever before; the incidence of hardness of hearing is not decreasing; "head colds" are universal experiences; tonsillectomies are every-day occurrences; and the business of selling patent "hair restorers," which do not even restore hope, was never more prosperous. Research in all of these fields is actively in progress, in ardent expectation of ultimate success in each. The failures of medicine and dentistry, to prevent various diseases, are due *honorably* to the same reason; to inherent difficulties that the most competent, devoted, and ingenious efforts

have *not yet* been able to surmount. Failure to attain agreement on the way or ways to prevent caries is a continuing challenge to each center of dental research, as well as to the dental profession as a whole—and to all who seek to promote the public welfare by perfecting the resources of oral health-service.

The Lancaster Press has been requested to hold the type of the second edition in anticipation of its use in a possible third edition.

WILLIAM J. GIES, *Secretary,*
Advisory Committee on
Research in Dental Caries.

SUMMARY

Provision must be made for the expansion of the work in physical research at the National Bureau of Standards. We now have twelve specifications which require regular testing of materials bought on the open market if these specifications are to have the importance desired. Such routine testing now demands the exclusive attention of one of our research associates at the Bureau, and, in order to keep the lists up to date, it is necessary to increase this service. The more time that is devoted to testing, the less opportunity there is for investigative research. This matter is mentioned at this time in order to emphasize the importance of making plans for expanding the budget appropriation for the work at the Bureau of Standards.

The sale of the first edition of "Dental Caries" has practically paid for the entire expense involved in this undertaking. The second edition of "Dental Caries" is limited to thirteen hundred copies. At $2 a copy, it is expected that the second edition also will pay for itself.

September 15, 1941, Edward G. Hampp was appointed and entered upon active duty as research associate in connection with the new fellowship being established for biologic research in dental diseases at the National Institute of Health. Dr. Hampp has received the degrees of D.D.S. and M.S., from Washington University, St. Louis, Mo. In 1936, he was awarded a three-year fellowship from the Carnegie Foundation. His work during that time was done at Washington University and at the University of Rochester in the departments of cytology and pathology, respectively. He spent the summer of 1939 in the Department of Bacteriology, University of Michigan Medical School. H. Trendley Dean, dental surgeon in the Division of Infectious Diseases at the National Institute of Health and member of the Research Commission, under whose direction the work will be done, visited Dr. Hampp in St. Louis. His approval was given before the selection was made. The Research Commission is convinced the work done by Dr. Hampp at the Institute will prove valuable to dentistry and thus once again justify the faith of the American Dental Association in the Commission's judgment.

M. D. HUFF, *Chairman,*
DANIEL F. LYNCH, *Secretary.*

Washington, D. C.
September 15, 1941.

COUNCIL ON DENTAL EDUCATION

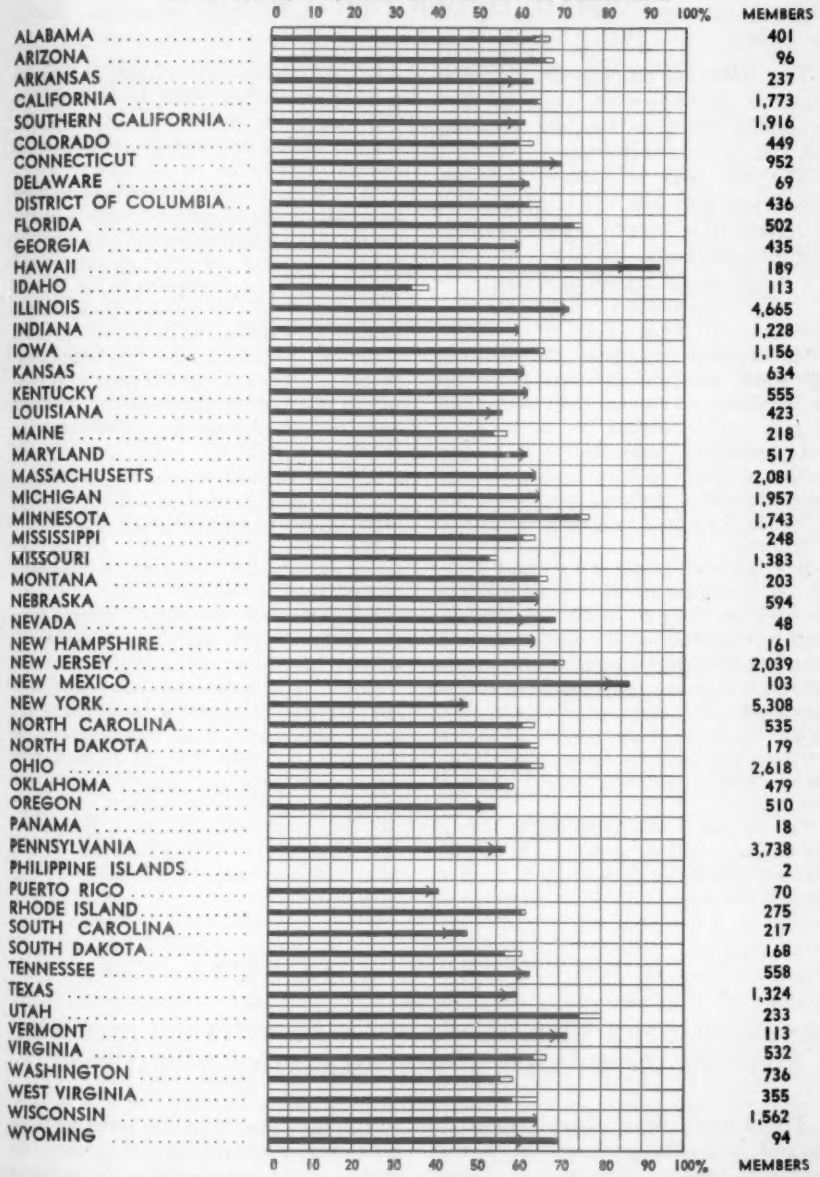
ENROLLMENT IN THE DENTAL SCHOOLS OF THE UNITED STATES

	Opening of Academic Year, September 1941 Subject to Change in Dental Students' Register for 1941					October 15, 1940 Dental Students' Register				
	Year					Year				
	1	2	3	4	Total	1	2	3	4	Total
Coll. of Physicians & Surgeons	46	40	42	34	162	43	43	35	30	151
U. of California	55	49	37	40	181	62	35	42	29	168
U. of S. California	145	105	85	50	385	119	85	57	77	338
Georgetown	66	44	47	43	200	56	44	42	41	183
Howard U.	30	25	18	17	90	24	22	15	11	72
Atlanta-Southern Dental Coll.	101	103	78	70	352	118	77	72	52	319
Loyola U. (Chicago)	60	85	73	55	273	92	75	58	44	269
Northwestern U.	96	58	64	84	302	72	62	70	91	295
U. of Illinois	58	62	56	50	226	61	67	50	46	224
Indiana U.	44	43	25	19	131	47	29	19	18	113
State U. of Iowa	40	35	40	18	133	37	41	18	16	112
U. of Louisville	54	40	54	37	185	47	50	39	41	177
Loyola U. (New Orleans)	42	35	37	43	157	45	38	43	28	154
U. of Maryland	98	75	86	63	322	86	85	64	75	310
Harvard U.	9*	38	36	34	117	40	40	33	33	146
Tufts College	83	74	68	57	282	74	64	56	63	257
U. of Detroit	15	22	23	32	92	22	24	32	28	106
U. of Michigan	69	39	46	32	186	42	49	32	35	158
U. of Minnesota	83	58	59	62	262	64	62	62	9	197
U. of Kansas City	63	85	61	55	264	91	67	53	29	240
St. Louis U.	82	56	40	58	236	54	30	58	25	167
Washington U.	45	16	21	25	107	16	22	24	28	90
Creighton U.	20	18	27	12	77	18	24	15	19	76
U. of Nebraska	32	14	15	12	73	12	14	11	11	48
Columbia U.	51	53	54	48	206	60	56	47	52	215
New York U.	149	148	138	126	561	157	142	129	123	551
U. of Buffalo	55	..	41	45	141	56	..	44	42	142
Ohio State U.	57	62	65	50	234	62	61	55	44	222
Western Reserve U.	60	36	43	33	172	41	42	34	44	161
N. Pacific College	80	54	58	39	231	55	55	37	24	171
Temple U.	118	111	92	136	457	116	92	135	96	439
U. of Pennsylvania	121	105	88	98	412	116	94	98	117	425
U. of Pittsburgh	96	90	74	74	334	96	82	73	46	297
Meharry Medical College	23	9	6	10	48	12	9	12	8	41
U. of Tennessee	28	26	29	27	110	33	29	28	15	105
Baylor U.	57	38	41	36	172	36	47	36	25	144
Texas Dental College	38	34	23	18	113	34	23	19	11	87
Medical College of Virginia	48	32	28	31	139	33	37	30	35	135
Marquette U.	75	53	60	58	246	56	55	64	40	215
TOTALS	2492	2070	1978	1831	8371	2305	1973	1841	1601	7720

*Entering class in Harvard School of Dental Medicine.

Membership Page

PERCENTAGE OF DUES RECEIVED IN CENTRAL OFFICE AS OF SEPTEMBER 30, 1941, AS COMPARED TO 1940 MEMBERSHIP



Percentage based on dental population figures supplied by the State Society Secretaries

1941 percentage. 1940 percentage.

COMMITTEE ON DENTAL LEGISLATION

MAIL ORDER DENTURES

THE following bill was introduced in Congress, September 17, 1941, by Representative Philip A. Traynor, Wilmington, Del., a licensed dentist.

The bill seeks to prevent improper dental practices and the circumvention of certain state laws by mail order denture manufacturers.

IN THE HOUSE OF REPRESENTATIVES
September 17, 1941
H. R. 5674

Mr. Traynor introduced the following bill; which was referred to the Committee on Interstate and Foreign Commerce.

A BILL

To protect the public health by the prevention of certain practices leading to dental disorders; and to prevent the circumvention of certain State laws regulating the practice of dentistry.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled, That it shall be unlawful, in the course of the conduct of a business of constructing or supplying dentures from casts or impressions sent through the mails or in interstate commerce, to use the mails or any instrumentality of interstate commerce for the purpose of sending or bringing into any State the laws of which prohibit—

(1) the taking of impressions or casts of the human mouth or teeth by a person not licensed under the laws of such State to practice dentistry;

(2) the construction or supply of dentures by a person other than, or without the authorization or prescription of, a person licensed under the laws of such State to practice dentistry; or

(3) the construction or supply of dentures from impressions or casts made by a person not licensed under the laws of such State to practice dentistry, any denture constructed from any cast or impression made by any person other than, or without the authorization or prescription of, a person licensed under the laws of the State into which such denture is sent or brought to practice dentistry, or any matter advertising or soliciting orders for any denture so constructed or so to be constructed.

SEC. 2. As used in this Act, the term—

(1) "Denture" means a set of artificial teeth, or any prosthetic dental appliance;

(2) "State" includes the District of Columbia and any Territory or possession of the United States; and

(3) "Interstate commerce" means commerce between any State and any place outside thereof.

SEC. 3. Any violation of any provision of this Act shall be punished by a fine of not more than $1,000 or by imprisonment for not more than one year, or by both such fine and imprisonment. Upon a second conviction of an offense punishable under this Act the punishment shall be a fine of not more than $5,000 or imprisonment for not more than five years, or both such fine and imprisonment.

RECENT COURT DECISIONS

DENTAL PRACTICE ACTS: REFERRING OF PATIENTS TO UNLICENSED PERSONS AS "FRAUD OR MISREPRESENTATION IN PRACTICE"

The Arizona State Dental Board instituted proceedings to revoke Batty's license to practice dentistry on charges that (1) he was physically incompetent to practice because he was suffering from pulmonary tuberculosis; (2) he was guilty of misrepresentation in securing his license, in that he represented him-

self at that time to be free from any malignant, infectious or contagious disease, whereas he was in fact suffering from pulmonary tuberculosis, and (3) he had been guilty of fraud in the practice of dentistry in that he had (a) practiced under a name not included in his license and (b) referred certain patients to a dental laboratory wherein dentistry was practiced by unlicensed persons. After notice and hearing, the board found that all charges had been sustained and, in effect, revoked his license. The dentist brought certiorari to the superior court, Maricopa County, which affirmed the order of the board, and he appealed to the Supreme Court of Arizona.

The dental practice act, said the Supreme Court, authorizes the board of dental examiners to revoke a license, among other things, for (a) physical or mental incompetency, (b) fraud or misrepresentation in securing a license and (c) fraud or misrepresentation in the practice of dentistry, all of which acts the dentist in this case was charged with committing. Proof of one charge at least was necessary to give the board jurisdiction to revoke his license.

The court concluded that the first charge against the dentist (that he was physically incompetent to practice dentistry because of pulmonary tuberculosis) had not been sustained by the evidence before the board. The court said that all that the evidence indicated was that at one time the dentist had drawn compensation for tuberculosis, but that the case had become arrested. A person with an arrested case of tuberculosis is of no more danger to a patient, as far as infection is concerned, than one who has never had the disease.

The court next determined that the second charge against the dentist (that he had been guilty of fraud or misrepresentation in securing his license to practice because at that time he was afflicted with tuberculosis) was also not sustained by the evidence before the board. The only evidence on this point, said the court, is that the dentist was examined by two reputable physicians just before securing his license and certified as free from active tuberculosis.

The court did not approve of the charge that the dentist was guilty of fraud and misrepresentation in the practice of his profession because he practiced under names not included in his license. The mere fact that the dentist sometimes spelled his name "Batty" and other times "Battie" was not evidence of fraud. The two names were pronounced the same and there was no evidence that he used two spellings for fraudulent purposes.

With respect to the charge that the dentist had committed fraud by referring the patient to an unlicensed person for dental operation, the court felt that the evidence was more substantial. It was proved that the dentist was present when a patient was directed to go to a dental laboratory to have denture impressions made. The dentist knew that the person who made the impression was not a licensed dentist. He subsequently met the patient at the dental laboratory when the plate was inserted and received $4 for the extractions that he had performed between the taking of the impressions and the insertion of the dentures.

Does this testimony, asked the court, if believed by the board, justify the conclusion that the dentist has committed fraud in his practice? The duty of the dentist is to advise the patient properly in all matters pertaining to his ailments, and this certainly includes advice as to who are properly qualified to assist in his treatment. Fraud may be committed by a failure to speak when the duty of speaking is imposed as much as by speaking falsely. A physician who knows that a patient of his is about to have surgical or medical work done by one who has

no legal right to do it fails in his duty if he does not advise the patient of the situation.

The court accordingly concluded that the board had acted within its jurisdiction and, in effect, affirmed the order of the board revoking the dentist's license to practice.—Batty *v.* Arizona State Dental Board, 112 P. (2d) 870 (Ariz. 1941).

DENTRL PRACTICE ACTS: SALE OF ARTIFICIAL TEETH NOT A "DENTAL OPERATION"

THE Indiana State Board of Dental Examiners sought to obtain an injunction against Sam Lepon, doing business as the Federal Dental Laboratory, on the ground that he was practicing dentistry without a license. The evidence indicated that the defendant maintained a display room where the customer could select from samples the kind of plates, bridges or artificial teeth desired. The customer was then asked to procure an impression of his mouth from a licensed dentist. This impression with the customer's order was then sent to Chicago, where the artificial teeth were manufactured. When completed, the teeth were returned to the laboratory, which delivered them to the customer. The customer paid both the laboratory and the dentist. In this way, the defendant had no direct physical contact with the mouth of the customer. There was no evidence as to who fitted or adjusted the teeth if such was required, so this could not be taken into consideration. On the basis of this evidence, the lower court refused to grant an injunction to the board of dental examiners and the board appealed to the Supreme Court of Indiana.

The only question presented, said the court, was whether the defendant's method of doing business and the advertising thereof were within the definition of the practice of dentistry contained in the dental practice act, the pertinent portions of which are:

> Any person shall be said to be practicing dentistry . . . who . . . advertises . . . that he can or will attempt to perform dental operations of any kind . . . or supplies artificial teeth as substitutes for natural teeth. . . . And, provided, further, that nothing in this act shall interfere with the performance of mechanical work on inanimate objects by any person employed in or operating a dental laboratory. . . .

The sale of artificial teeth is not a dental operation. Their manufacture from inanimate impressions is permitted by the section quoted above, which would be ineffective if they could not be sold.

The dental board concedes, continued the court, that an unlicensed laboratory may sell to a licensed dentist, but asks us to read into the statute a prohibition against sale to the public. The statute does not forbid such a sale even though the sale of artificial teeth which do not fit may be detrimental to the health of the user. An amendment to forbid such a sale may be desirable, but the remedy lies with the legislature and not the courts. Upon the facts in this case, the defendant is not violating a prohibition of the statute. Judgment affirmed.—State *ex rel.* Kennedy *et al. v.* Lepon. 36 N.E. 2d. 276 (Ind. 1941).

NEWS ITEMS

THE ARMY DENTAL CORPS*

EXPANSION of the Army has meant the construction of forty-six camps for 10,000 or more troops. Some of these camps have nearly 75,000 troops. There are more than seventy other camps and air corps stations. There are one or more hospitals in every camp, the number depending on the strength of the command. Twenty of these hospitals provide for from 1,000 to 2,000 patients. There are fourteen general hospitals. Exclusive of dental clinics in hospitals, there are more than 100 dental clinics, each containing from ten to twenty-five operating rooms and other necessary installations. Sixty dental laboratories, serving all the many dental clinics, have been provided. Each camp has a senior dental officer in charge of the entire dental service of the camp. Every hospital has a dental service with most carefully selected personnel, dental officers experienced in hospital dental service.

With the combat organizations, we have the dental service for duty with the field forces. There are two cavalry divisions, twenty-seven infantry divisions and four armored divisions. Many other miscellaneous tactical units are provided in large numbers, such as general service engineer regiments, antiaircraft regiments, mountain artillery and chemical warfare units. Each of these organizations has its quota of dental officers, who serve with the regiments during the training period and in combat.

The equipment for all these installations and units has been carefully selected. Hospitals and clinics are supplied with excellent chairs, units, lights, x-ray machines, electric sterilizers and all other modern facilities of recent design, together with instruments and supplies common in all modern dental offices today. The field equipment is entirely new. Instead of the old field dental outfits of World War days, which consisted of five pieces of baggage, the new field outfit is all contained in one trunk, the total weight of which is 165 pounds. The dental laboratories are complete in every detail and provide facilities for replacements of the highest quality. The finest types of partial and full dentures, using gold or vitallium when necessary, and vulcanite or acrylic denture base material as indicated, are available in the dental service.

The training of dental personnel is important. Five general hospitals provide facilities for the special training of selected reserve officers for duty as chief clinicians, prosthetists and oral surgeons for assignment to hospitals and clinics. The Army Dental School is the dominating influence in the professional training of the dental officers. The need for this training becomes apparent when one recognizes the demand for the complete development of the policy for dental service. Standard methods for modern dentistry, as taught at the Army Dental School and applied in the general hospitals and clinics, will rule. Dental service for military personnel places a great responsibility upon both those who formulate the policy and the many who make that policy effective. Regulations provide that:

> Dental attendance for all military personnel will be confined to the treatment of emergency cases, infectious conditions, and the restoration of carious teeth with amalgam, silicate, or cement fillings. . . . Replacement of missing teeth will not be made except when teeth are damaged or lost in the performance of duty, while engaged in

*From the office of the Surgeon General of the Army.

athletic games, or as a necessary part of treatment. Such replacements will be the standard type of partial or full dentures provided for military personnel.

To carry out such a program for the large army now mobilizing, it is necessary to secure many hundreds of skilled dental assistants, laboratory technicians and dental hygienists. Chair assistants and laboratory technicians require special training for the exacting service demanded. Men with suitable qualifications are assigned to the dental service for training when inducted and are sent to the medical replacement centers. Those with acceptable laboratory experience are then sent to the special training schools for dental technicians at the general hospitals. Upon completion of their training, they are assigned to the camp dental laboratories. Of course, they will work under the immediate supervision of a dental officer especially trained in Army prosthesis. In accordance with the dental policy, a dental officer is in charge of the dental laboratory. Provisions have been made for an allowance of 148 dental hygienists, all of whom must have civil service status. X-ray technicians for each dental clinic will be trained at the general hospitals and the Army Dental School.

We not only must furnish adequate clinical facilities in every camp, but we must also provide the training of dental personnel in the tactical or combat units. Because of the different functions and responsibilities of the dental personnel in the camps, we have provided War Department directives which definitely separate the two groups, those in the hospitals and clinics from those with the combat units. While in camps, the regimental dental officers limit their actual dental service to daily sick call, treatment of limited emergency cases and examination of all the men of the unit. The remainder of their time is devoted to training in a variety of medical and tactical subjects. This is revealed in the following quotation taken from instructions issued to dental officers:

> Dental service at regimental and separate battalion dispensaries and aid stations will consist of emergency service and dental surveys in the tactical units to which the dispensaries are attached and will be provided by dental officers attached to the regiment or separate battalion. Definitive treatment, serious extractions, and treatment which demands extensive dental equipment will be provided in camp or hospital dental clinics and will be under the control of the Corps Area Service Command. Dental officers of the tactical units will receive training in medical tactics as auxiliary medical officers and in emergency treatment of jaw casualties in their respective units. Technical instruction in more extensive definitive dental treatment will be provided in the camp or hospital dental clinics. Training activities in medical tactics and functions of the regimental and separate battalions will be under the direction of the division or unit surgeon. Technical training in camp or hospital dental clinics will be under the direction of the camp or station surgeon.

The scope of the dental service in camps, clinics and hospitals will meet the most exacting demands for any military unit and should receive the hearty commendation of the dental profession. The end-results can be measured by the honest efforts of the thousands of dentists who are called to serve.

Unlike the days of 1917-1919, the dental service is provided with an efficient administrative set up. A large and busy group constitute the dental division in the Surgeon General's office. This office develops and coordinates all dental activities in the Army, and it is here that the policy for the dental service is developed. Matters of direct policy are all subject to approval by the Surgeon General and the War Department. The activities of the dental division are also concerned with the problems connected with personnel, professional matters, supply, clinics, plans and training, statistics, etc. Contact with the dental serv-

ice in the field is made through the corps area surgeons to the corps area dental surgeons. The former are responsible for the dental service in their respective areas. Through the camp surgeons, they contact the dental services in their camps, the camp dental surgeons making the dental policy effective in every installation. This assures the dental supervision so essential to guarantee an adequate and efficient dental service throughout the entire Army. From the War Department on down to the smallest dental clinic, all the possibilities for the fullest development of the dental service depend on the sound development of dental activities by the senior dental officers in charge. The plans for the dental clinics have all been developed by the dental division in the Surgeon General's office. The selection of supplies and equipment has been made by the same agency. The plans for training have been developed through the coordinated efforts of the Army Dental School, the dental division and related agencies within the Surgeon General's office. The selection of personnel for key positions has been made by the personnel section of the dental division, through the assistance of the corps area dental officers. The preparation of many articles for publication has been accomplished by experienced dental officers in special fields. Lectures, material for clinics and demonstrations, photographs and lantern slides have been prepared for presentation in connection with the many training courses provided for training centers and special schools. In all these activities, there has always been evidenced on the part of every dental officer a great desire to make the entire dental program a complete and efficient one, in keeping with our expanding dental service.

CHICAGO DENTAL SOCIETY ESSAY AWARD

CASH prizes totaling $500 for the best two essays on dental subjects are offered by the Chicago Dental Society, the winners to present their essays at the opening general session of the 1942 Midwinter Meeting. The first award offered is $300, the second, $200. The meeting will be held at the Palmer House February 23, 24, 25 and 26. The purpose of this competition is to encourage scientific research and investigation in dentistry.

RULES

Eligibility to Apply for Participation.—Any American Dental Association member or other person of scientific attainment affiliated with a recognized institution in the dental field shall be eligible to apply for permission to enter the competition. The Chicago Dental Society, through its duly appointed committe, shall pass upon the qualifications of applicants. Application forms can be secured on written request to the Chicago Dental Society, 30 North Michigan Ave., Chicago.

Character of Essays.—Each essay submitted must represent an original investigation and contain some significant new material of value to dentistry.

Presentation of Winning Essays.—The authors of the two winning essays will be invited to present them at the opening general session of the Midwinter Meeting, it being understood that, irrespective of the number of coauthors or collaborators, only one shall be invited. The expenses of the winner will be paid by the Chicago Dental Society on the same basis as regularly invited Midwinter Meeting essayists.

Ownership and Publication.—The two winning essays are to become the property of the Chicago Dental Society and will be published in the most advan-

tageous manner. All other essays will be returned to their respective authors.

Final Date for Acceptance of Entries and Future Policy.—No essays will be considered for this competition unless submitted to the society on or before December 31, 1941. It is proposed that this award shall be offered annually hereafter. Therefore, beginning with the 1943 competition, essays must be submitted not later than the November 1 preceding.

All inquiries pertaining to the competition should be addressed to the society at its offices, 30 North Michigan Ave., Chicago.

PERIODONTIA RESEARCH

THE Periodontia Department of New York University College of Dentistry cordially invites each investigator in the field of periodontal disease who has published a paper or papers containing the results of either laboratory research or clinical observations which bear directly or indirectly upon the etiology or treatment of periodontal disease in man or animals to (1) submit a summary of those of his papers which he deems his basic contributions to this field and which he considers to be in agreement with his present thinking, or with his experience and thinking up to a time specified by him; (2) cite the time and place of appearance of the paper or papers that contain such basic findings, and (3) enclose any additional statement that may seem necessary to him to so amend the foregoing statements that they more accurately reflect his present opinions.

These summaries will, therefore, contain only present opinions, rather than the total results of each worker's endeavors, which may include errors, duplications, altered viewpoints, etc. The compilations of such summaries, which will contain the essence of each investigator's present thinking, will then be published in book form and will, therefore, not be a series of abstracts of papers on either the etiology or the treatment of periodontal disease.

The summary should be in our hands not later than March 15, 1942.

New York University,
College of Dentistry,
209 E. 23d St., New York, N. Y.

THE AMERICAN BOARD OF ORTHODONTICS

CERTIFICATES of qualification were granted to the following orthodontists by the American Board of Orthodontics in 1941:

F. Copeland Shelden, Kansas City, Mo.
E. Allen Bishop, Seattle, Wash.
Louis Braun, Detroit, Mich.
James A. Burrill, Chicago, Ill.
Burl Francis Dewel, Evanston, Ill.
P. M. Dunn, Minneapolis, Minn.
Meyer Eggnatz, Baltimore, Md.
Bercu Fischer, New York, N. Y.
James W. Ford, Chicago, Ill.
Samuel Hemley, New York, N. Y.
William C. Keller, New York, N. Y.
Ruben E. Olson, Wichita, Kan.
William A. Pearson, Norfolk, Va.
Franklin A. Squires, White Plains, N. Y.
Alexander Sved, New York, N. Y.
R. F. Thompson, Washington, D. C.
M. Bagley Walker, Norfolk, Va.
Willis Hull Murphey, Fort Worth, Texas.

BERNARD G. DEVRIES, *Secretary*,
Medical Arts Bldg.,
Minneapolis, Minn.

OBITUARIES

TRIM HOUSTON, D.D.S.
(1875-1941)

TRIM HOUSTON died June 1, 1941, at the Physicians and Surgeons Hospital, Corsicana, Texas, after a major operation. He was born at Petersburg, Tenn., July 15, 1875. The family moved to Texas when he was about 9 years of age. His father, B. F. Houston, was one of the pioneer eye, ear, nose and throat specialists of Texas.

Dr. Houston received his dental education at the University of Tennessee, which was at that time in Nashville. He graduated in 1895 and returned to Corsicana, where he spent his entire professional life. At various times, he took special postgraduate work at Vanderbilt and at Northwestern University. He was a member of the Sixth District, Texas Dental Society and the American Dental Association; a former chairman of the membership committee of the Texas Dental Society and, at the time of his death, a member of the Twelfth District Membership Committee of the American Dental Association. In 1927, he was chosen president of the Texas Dental Society. From 1934 through 1936, he served as chairman of the Texas Dental Society Membership Committee, laboring diligently at his task and being rewarded by an increase in membership of more than 500, giving the society an impetus the effects of which are still evident.

Aside from his devotion to his family and love of his profession, Dr. Houston's chief interest was in the dental welfare of the children of the I O O F Home, located in Corsicana. He took over this work in 1907 and, throughout the years, it was largely a labor of love. He was interested in welfare work and devised many original methods of care, administration and diet in the I O O F institution, in the meantime carrying on a lively correspondence with dentists throughout the world engaged in similar work.

At the time of his death, he was busily engaged in compiling some records of the institution for the Committee on Economics of the American Dental Association, this institution being selected as one of the three in the United States

TRIM HOUSTON

that had always had a sound dental program.

Organized dentistry had no stauncher advocate, and, in the course of forty-five years of a busy and active practice, he lived to see some of his most cherished dreams come true.

Dr. Houston is survived by his wife and four daughters, Mrs. R. E. O'Berry, Mrs. C. N. Weakly, Mrs. Nat Burr and Miss Edith Houston.

HARRY EZELL.

DEATHS

ADAMS, FRANKLIN S., Waterville, Kan.; Baltimore College of Dental Surgery, 1899; died June 16.

ALEXANDER, WILLIAM ALVA, Litchfield, Ill.; Indiana University School of Dentistry, 1888; died July 24; aged 76.

BARD, ALEXANDER E., Tucson, Ariz.; University of Pittsburgh, School of Dentistry, 1903; died August 20; aged 61.

BEHRENS, F. C., Gillespie, Ill.; Missouri Dental College, 1901; died recently; aged 62.

BROWNING, ERNEST W., Lenox, Iowa; State University of Iowa College of Dentistry, 1900; died July 16; aged 69.

BUCKLEY, MICHAEL J., Chicago, Ill.; Northwestern University Dental School, 1908; died July 31; aged 64.

DIXON, FRANK L., Rock Valley, Iowa; Northwestern University Dental School, 1905; died July 20; aged 58.

DREW, ERNEST DANIEL, Livingston, Texas; died July 28; aged 67.

DUNHAM, O. A., Manchester, Iowa; State University of Iowa College of Dentistry, 1886; died June 9; aged 88.

ECKHARDT, GEORGE F., Austin, Texas; Tulane University School of Dentistry, 1902; died September 2; aged 62.

GREENBERG, ROBERT W., New York, N. Y.; New York College of Dentistry, 1905; died July 29.

HARMKE, CARL W., Chicago, Ill.; Northwestern University Dental School, 1924; died July 6; aged 38.

HARRIS, SED A., Snyder, Texas; Northwestern University Dental School, 1902; died August 2.

HOCKABOUT, WILLIAM, Gravity, Iowa; died June 6; aged 72.

HOLMES, JOHN M., Pittsburgh, Pa.; University of Pittsburgh School of Dentistry, 1900; died October 6.

HUMPHREY, GLENN H., Ames, Iowa; State University of Iowa College of Dentistry, 1914; died June 27; aged 49.

JERICHO, SMITH DAVIS, Mount Pleasant, Iowa, formerly of Moline, Ill.; Northwestern University Dental School, 1916; died June 3; aged 49.

LOCKHART, JAMES L., San Antonio, Texas; Baylor University College of Dentistry, 1925; died August 29; aged 40.

LOCKE, SEON, Springer, N. M.; Western Dental College, 1898; died September 10.

MCLEAN, RICHARD W., Bloomington, Ill.; Chicago College of Dental Surgery, 1917; died August 14; aged 48.

MCMANUS, HENRY, Washington, D. C., formerly of Hartford, Conn.; Temple University School of Dentistry, 1890; died August 10; aged 73.

MAHONEY, EDWARD, Little Rock, Ark.; Vanderbilt University School of Dentistry, 1918; died April 13.

MALKIN, BARNEY E., Youngstown, Ohio; Western Reserve University School of Dentistry, 1918; died March 21.

RIESTER, PAUL H., Pittsburgh, Pa.; University of Pittsburgh Dental School, 1926; died August 20; aged 37.

SHAW, W. A., Belleville, Kan.; died recently; aged 64.

STEWART, LAFAYETTE A., Sullivan, Ind.; Indiana University School of Dentistry, 1888; died August 2; aged 77.

STONE, D., Sandersville, Ga.; Southern Dental College, 1915; died June 18; aged 55.

SWANSON, EDWARD, Captain, Dental Corps, Portland, Ore.; North Pacific College of Oregon, 1933; died August 3; aged 32.

TITUS, HARRY W., Eugene, Ore.; North Pacific College of Oregon School of Dentistry, 1917; died July 29; aged 47.

VETTER, CHARLES, New York, N. Y.; New York College of Dentistry, 1896; died August 28.

WARD, GRIFFY GOLDING, Oelwein, Iowa; University of Michigan School of Dentistry, 1907; died July 7; aged 57.

WESTFALL, GEORGE, Carlsbad, N. M.; Washington University School of Dentistry, 1917; died August 28.

WHEELER, IRVING L., Traverse City, Mich.; University of Michigan School of Dentistry, 1905; died June 21.

YOUNG, JOSEPH LEROY, Muscatine, Iowa; State University of Iowa College of Dentistry, 1905; died July 8; aged 60.

INCIDENTS OF PRACTICE

Operation for Removal of Radicular Cyst

By Reubin Seldin, D.D.S., New York, N. Y.

History.—A woman aged 35, white, in good health, complained of a small swelling of the left mandible accompanied by slight tenderness. A lateral roentgenogram revealed a large cystic area extending from the left second bicuspid to the third molar area. The apices of the second bicuspid and second molar area were in close proximity to the cyst. These two teeth were still vital, as were the other teeth on this side.

Operation.—Anesthesia of the left mandible was induced by an inferior alveolar block with 2.5 cc. of monocaine, supplemented by buccal infiltration.

Fig. 1.—Preoperative film, taken February 6, 1939.

Fig. 2.—Postoperative film, taken March 24, 1939.

A vertical incision from the interproximal

Fig. 3.—Postoperative film, taken February 29, 1940.

space anteriorly from the first bicuspid and down to the mucobuccal fold, and a horizontal incision from the anterior aspect of the first bicuspid along the necks of the teeth to the third molar, were made. A mucoperiosteal flap was deflected and the buccal surface of the mandible exposed. Extreme care was necessary to avoid injury to the mental nerve.

An opening in the bone was made with hand chisels and rongeurs, directly laterally from the cyst, between and below the second bicuspid and second molar teeth. The opening was seven-eighths inch in diameter.

The cyst was opened and its contents aspirated. The deflated cyst was then carefully pealed out *in toto*. Special care was taken to avoid injury to the apices of the bicuspid and molar involved and to the contents of the inferior alveolar canal which lay exposed just under the cyst wall.

The bone cavity left by the removal of the cyst was painted with a 1 per cent aqueous solution of acriviolet. The flap was then sutured in place.

Outcome.—Two days later, there was moderate swelling, with tenderness, but no pain. There was some numbness in the region of the mental foramen and of the lip. The sutures were removed five days after operation. Postoperative x-ray films over a period of one year showed the cyst cavity filling in. The last x-ray film showed almost complete regeneration of the bone destroyed by the cyst. The numbness of the lip disappeared after several months. All teeth were found to be vital one year after operation.

122 East Forty-Second Street.

Conserving Mercury

By LOUIS M. FLEISCH, D.D.S., Topeka, Kan.

MERCURY has been placed on the priority list of defense materials and should be conserved if the best interests of national defense are to be served.

A few years ago, I read an article concerning the habit many dentists have of expressing excess mercury from amalgam on to the floor. The article suggested that this practice could be dangerous to the health of the dentist. The accumulated mercury on the floor gradually assumes the form of dust and this can be inhaled. The theory was that the continuous exposure to the mercury dust might be harmful to the health of the dentist.

This article impressed me to the extent that thereafter I had my assistant express the excess mercury from amalgam into the lavatory. I was surprised at the amount of mercury that accumulated in the trap. The last time that the trap was cleaned, I recovered 1 pound of scrap mercury, approximately a two-year accumulation. It would be well to have the janitor remove the trap and clean it and then remove the trap at six-month intervals to remove scrap mercury. Conserving mercury in this manner can be beneficial to health, purse and nation.

504 Central Building.

Supernumerary Molars

By EDWIN V. ERBE, D.M.D., Waterbury, Conn.

A MAN, aged 35, had a supernumerary tooth on the upper right side (Fig. 1),

posteriorly from the third molar, and, in the lower jaw on the same side, the third molar was half again as large as the first or second molars and had three roots. (Fig. 2.) Whether the latter case resulted from fusion

Fig. 1.—Supernumerary tooth.

Fig. 2.—Third molar showing three roots.

of a normal third molar with a supernumerary tooth, I do not know. The third molars on the opposite side were normal.

Waterbury National Bank Building.

Abrasive Markings on Teeth

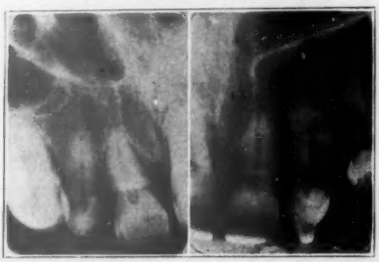
By JOHN A. HIGGINS, D.D.S., Blue Island, Ill.

PATIENTS sometimes present abrasive markings on one or more teeth, usually in the buccogingival area of the lower cuspids and bicuspids, but occasionally on the buccogingival surfaces of other teeth.

Upon inquiry, the patient usually states that he uses a hard bristle toothbrush with some form of toothpaste, toothpowder, soap, sodium bicarbonate (baking soda) mouthwashes and salt or salt dissolved in water. The brushing in all cases is carried out with a horizontal motion.

The possibility that there are abrasive ingredients in toothpaste, toothpowder, etc., is recognized, but to find abrasive markings on teeth cleaned with the cleansing ingredients dissolved in water or other liquid vehicle meant that the toothpowders or toothpastes were not alone at fault.

A patient whose mind was set on the extraction of his teeth was finally convinced that he could retain his own teeth for several years at least, and the teeth were scaled and cleaned. The patient was now instructed to get an extra hard bristle brush and use it with a liquid mouthwash or salt dissolved in water. Instructions were given to brush



Abrasive markings on teeth from brushing agents and technic.

the anterior teeth, especially, with a horizontal movement and the posterior teeth with a vertical movement; i.e., brushing down on the upper teeth and up on the lower teeth. After eight years of observation, the anterior teeth were roentgenographed and extracted. The roentgenograms show marked abrasion. At the same time, the posterior teeth showed no noticeable changes.

This would now seem to indicate that the type of toothbrushes used and the direction of brushing has much to do with tooth abrasion. Toothpaste, toothpowders or other cleansing agents play only a minor part in tooth abrasion if proper directions for brushing the teeth are followed.

13037 Western Avenue.

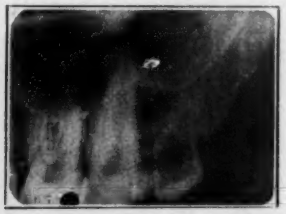
Third Molar Devoid of Dentin and Cementum

By C. M. STEBNER, D.D.S., Laramie, Wyo.

IN the regular routine of bitewing examination of a man, aged 21, I noticed an abnormal condition of the right third molar. Further x-ray examination of the area proved that there was no dentin or cementum present. The tooth appeared normal in comparison with the other molars. There

were no breaks in the enamel cap and the tooth appeared to be firm in its socket.

Conditions similar to this have been described as "pink tooth," but little reference to such conditions can be found. I have been informed that a tumorous condition had probably destroyed all of the body of the tooth, leaving only the enamel intact. In the accompanying roentgenogram, the



Third molar devoid of dentin and cementum.

denser area of alveolar bone in the outline of what might have been the root is evident. Apparently, under the enamel cap, there is only pulpal or tumorous tissue.

207 Roach Building.

Upper Supernumerary Unerupted Central Incisors: Report of Case

By MATTHEW LOZIER, B.S., D.D.S., New York, N. Y.

A GIRL, aged 8, with both of the upper permanent central incisors apparently missing, gave a history of loss of the upper deciduous central incisors about a year and a half previously.

Figure 1 shows both permanent central incisors, with almost fully developed roots, completely unerupted and overlapping the roots of the respective lateral incisors. The permanent central incisors are in turn partly and respectively overlapped from the buccal aspect by two supernumerary teeth with only partly formed roots. The supernumerary teeth, which are shaped like central incisors, are found in a somewhat more mesial position in relation to the permanent central incisors.

Under procaine anesthesia, the supernumerary teeth were removed, with care not to disturb the permanent central incisors, which were apparently prevented from normal eruption by the overlapping of the supernumeraries.

Further corrective treatment by orthodontic means may be instituted at some later time.

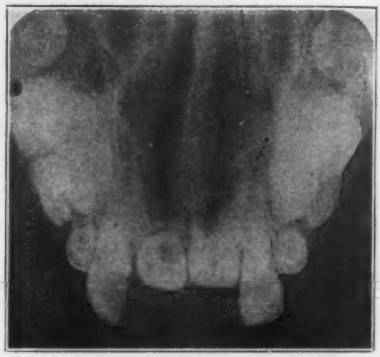


Figure 1.

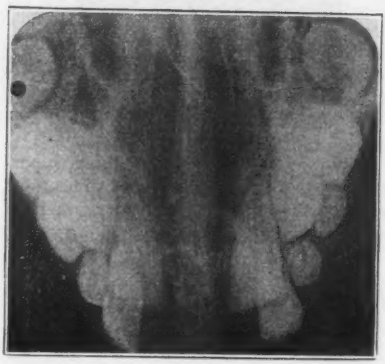


Figure 2.

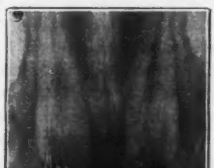


Figure 3.

Figure 2 shows the condition immediately after operation; Figure 3, a year later.

369 East One Hundred Forty-Ninth Street.

QUESTIONS AND ANSWERS

The answers here published have been prepared by competent authorities, but do not, however, represent the opinion of the American Dental Association as an organization or of any other official bodies unless specifically stated in the reply.

DENS-IN-DENTE

To the Editor: A boy, aged 10, developed an abscess above the lateral incisor, which was partly erupted. There was no pain and no soreness of the tooth, but considerable swelling. I lanced the abscess, getting considerable pus, and made a roentgenogram (Fig. 1, A) the third day, then waited ten days. The condition seemed to be clearing up. I took a second roentgenogram (Fig. 1, B), waited two weeks, and then took a third roentgenogram (Fig. 1, C). Tooth tests showed the tooth to be vital, but the mesial one-fourth of the tooth was discolored.

A B C

Figure 1.

Roentgenograms showed the fusing of an extra tooth. What caused the abscess, and what can we expect from this?

E. H. Hickman, D.D.S., Arcola, Ill.

Answer

The case submitted by Dr. Hickman is an unusual and interesting one. The roentgenograms indicate that the anomaly is a fusion of a normal lateral incisor and a supernumerary tooth which has a deep invagination of enamel on the lingual aspect. Teeth with such abnormally deep pits lined with enamel are called *dens-in-dente* (tooth within a tooth). Often, these deep lingual pits are not completely closed, a minute opening being left from the surface of the tooth into the pulp chamber. In such cases, the pulp of the tooth becomes infected and dies shortly after the tooth erupts. Apparently, that is what happened in this case. A minute exposure through the lingual pit of the supernumerary tooth resulted in infection of the pulp and an acute alveolar abscess developed. As the pulp of the normal lateral incisor was not affected, it remained vital and responded normally to pulpal tests.

The treatment of choice seems to be to attempt root-canal therapy in the supernumerary tooth with the hope that the fused tooth may be held in position, at least until the patient is old enough to have a fixed bridge placed. As the two teeth are firmly fused together, removal of the supernumerary tooth alone

is impractical. If root-canal therapy fails, extraction of the fused tooth is indicated, followed by construction of some sort of an appliance to preserve the space and answer the demand of esthetics.

M. H.

IRRITATION CAUSED BY PULP STONE

To the Editor: On March 27, 1941, a young married man, of about 30 years, came to my office. The right side of his face was quite swollen and he was suffering severe pain. Following is the history of the case, as he gave it to me.

Three weeks previously to his coming to my office, his lower right jaw began to swell, accompanied by pain. He went to a dentist, who, after examination and making an x-ray picture of the area, referred the patient to a physician, as he thought this condition belonged to the medical field. He went to a physician, who suggested hot compresses and prescribed a narcotic to relieve the pain. The condition became more aggravated, so the physician suggested that the patient see an eye specialist, as the trouble might be caused by an eye condition.

The eye specialist told him that his eyes were perfect, and sent him to a radiologist to have a lateral x-ray film taken. The patient went to an x-ray specialist, had a roentgenogram made and was referred back to the physician for diagnosis. The patient continued to suffer severe pain and the swelling continued. The physician lanced the outside of the cheek, but drew nothing but blood; and the narcotics were continued. All this occurred within a period of about three weeks. When the patient asked what the final outcome might be, the physician admitted that there was nothing more he could do for the patient. Then the patient consulted me.

My temporary clinical diagnosis pointed to the lower right second molar tooth. After taking a number of x-ray films of that area, I noticed a large pulp stone in the chamber of the tooth and it seemed to me that there might be a possible rarefaction of the distal root of the molar. The swelling on the lower jaw was very hard, and the tissues inside, between the cheek and the bone, were soft, and looked to me as if an incision on that area might bring pus. I incised the soft tissue and drew quite a bit of pus and the patient was relieved of pain at once. I then placed a drain of iodoform gauze in the opening and instructed the patient to use an ice bag externally, alternating one and a half hours on and one-half hour off throughout the day until bed time. I also prescribed a hot mouthwash. I saw the patient daily for a period of two weeks, changing the drain each day, and I advised that the ice treatment and the hot mouthwash be used as often as possible. The patient was getting better each day, the swelling becoming less, and the pain disappeared at the beginning of my treatment. The opening which I had made—about one and a half inches in length—kept closing up every day, and within two weeks was entirely closed.

I saw the patient a number of times after that. The swelling had disappeared entirely and the face was normal. I took two x-ray pictures of the area again (Figs. 1 and 2). The last one, April 29, 1941 (Fig. 2), looks normal except for the pulp stone. I saw the patient last on May 17, 1941, and everything seemed normal.

Figure 1.

Figure 2.

My diagnosis is this: I believe that the irritation produced by the pulp stone upon the pulp caused rarefaction of the distal root, and this in turn caused the whole trouble. I also believe that the tooth should be extracted.

Will you kindly give me your opinion of the case?

David S. Robinson, D.D.S.,
Newburgh, N. Y.

ANSWER

Kronfeld states ("Histopathology of the Teeth and Their Supporting Structures," second edition, page 94) that pulp stones never cause pulp inflammation, and suggests that, in clinical practice, it is wise to study and analyze all circumstances most carefully and rule out all other possible causes for pain and inflammation before condemning pulp stones. Pulp stones can be found in such a high percentage of adult teeth which show no sign of trouble that it would indeed be rare to have the pathologic condition described in the case above result from a pulp stone.

Before advising extraction of any teeth, it would be worth while to determine the reaction of the pulp of the second and third molars to thermal and electrical shock. If either tooth fails to respond to the pulp test, extraction is probably indicated, although root canal therapy might be attempted. If both teeth respond in normal manner to all tests, the case should be studied further. Lateral head plates should be obtained and careful examination be made of the tissues involved. The symptoms described above could be produced by an acute alveolar abscess in the lower right molar region following death of the pulp (because of trauma or some other condition) which had been allowed to localize. Incision and drainage in such cases result in satisfactory healing. Apparently, this treatment gave satisfactory results for Dr. Robinson, but the condition might recur if the cause was not removed.

M. H.

PYORRHEA ALVEOLARIS

To the Editor: What is the cause of pyorrhea alveolaris? In a fairly well-advanced case with recession of the jawbone, but with no dental or gingival symptoms, is there any local treatment or general treatment that might arrest the progress of the disease? Are calcium and vitamin D of any avail?

M. D.

ANSWER

As a rule, pyorrhea alveolaris is caused by gingival irritation, either mechanical or bacterial (usually both) and, in some instances, by excessive occlusal stress—in a word, oral sepsis and loss of occlusal balance. In some cases, and possibly to some extent in all, a systemic complication is present. In most instances, this acts chiefly as a predisposing factor, which can usually be overcome by proper attention to local conditions, as a result of which the local irritating factors are kept under control.

Treatment in these cases is almost entirely local. Without it, no amount of medication or dietetic measures will be of avail, though it is desirable that they be considered, as should a careful physical examination. In advanced cases, such as the one referred to, in which there is excessive loss of alveolar support, surgical measures are indicated, notwithstanding the statement to the contrary appearing in the *Journal of the American Medical Association,* June 14, 1941.

The absence of gingival symptoms referred to in this case is a matter of no real significance. The important thing is the loss of alveolar support and the probable creation of deep pockets. Elimination of these pockets by resection, establishment of oral hygiene and balancing of occlusion would appear to be the proper treatment. If the teeth are reasonably firm, the prognosis following such treatment would be good. Attention should be given to diet and to such other treatment as might be indicated by a physical examination. The important thing, however, in this case, as in all

cases of pyorrhea, is the local treatment. When this is carefully carried out and all local factors are taken into consideration, the results, as a rule, are highly satisfactory.

Arthur H. Merritt, New York, N. Y.

CONTROL OF HEMORRHAGE

To the Editor: We have recently had a hemophiliac as a patient. Following tooth extraction, there was considerable hemorrhage. Pressure, adrenalin chloride, thromboplastin and Monsel's salt were applied, with unfavorable results. It finally occurred to me that since human muscle is used to control hemorrhage in brain surgery, it may well be used in oral surgery. This was done by suturing a fresh piece of muscle, obtained from the operating room during a laparotomy, in the tooth socket. Hemostasis resulted. The result was very interesting and calls for further experimentation. I am well aware that muscle has been used, but the actual use of fresh human tissue is something new to me. The literature available to me is void of such a procedure. Can you enlighten me in any way regarding it?

Alfred T. Joseph, D.M.D.,
Boston, Mass.

ANSWER

The procedure of "muscle stamping" has been carried on for many years, and, although it is a procedure used rather frequently in persistent bleeding, there is little in the literature on the subject. The prompt action taken in an emergency such as existed in this particular case is commendable, but it is doubtful whether the procedure could be adopted for general use because of the danger of transmitting infection from one patient to another. Years ago, surgeons sometimes saturated a piece of cotton with their own blood and placed it over a wound, but they received much criticism for their heroic efforts.

Warbasse writes, in his book on surgical treatment, printed in 1918, "The local application of muscle, fascia, or fat will check bleeding in parenchymatous organs. Muscle is not effective [meaning that muscle of itself is not effective]. Bleeding from the liver, kidney, spleen or prostate is quickly checked by snipping off a piece of muscle in the adjacent wound and pressing it upon the bleeding surface. The transplanted tissue may be sewed in place to cover the bleeding area.

"In operating on the brain, bleeding often may be controlled by snipping off a bit of temporal or occipital muscle and placing it on the bleeding point."

Under the heading "blood serum or blood," Warbasse states: "The use of blood serum of another person applied at the bleeding point hastens coagulation.

"The surgeon in emergency may test this in the presence of a hemophilic wound, by making a small wound in himself and allowing the blood to drop into the wound of the patient. The cessation of bleeding is marvelously prompt. In hemorrhage of the new born and hemophilia these expedients are most useful."

"Muscle stamping" is done by using muscle from the patient. A publication on this subject would be most interesting, especially if it were to include the history of the procedure, which undoubtedly goes back to "folklore medicine" and might include the story of why people place beef on a black eye, etc. The scientific explanation would be most enlightening.

It is doubtful whether the articles printed on the subject will appear, in detail, in the books and periodicals of the average medical libraries.

C. W. S.

CURRENT LITERATURE

Osteitis Fibrosa Localisata

By John L. Bremer

It has been shown that osteitis fibrosa may be caused by a long-continued excess of estrogen (female sex hormone), probably acting through the parathyroid glands. An excess of estrogen that is soon reduced sufficiently to permit renewed normal growth may produce the local cyst. In this case, a small portion of mesenchyme, which indicates the initial stage of the fibrosis, is cut off within the bone and there undergoes any of the changes found in normal histogenesis, especially those leading to fibrous tissue and cartilage. This condition was reproduced experimentally. Such an enclosed mass might be expected to enlarge, erode vessels, degenerate and liquefy; giant cells might cluster around it as around a foreign body; or it might remain dormant for years. As an unusual variant of this more solid type, the true bone cyst could result from the rare combination of a temporary excess of estrogen or parathyroid activity with chronic internal pressure in a neighboring joint or bursa and the resultant entry of a diverticulum of either into the bone. Attempts to produce this have not been successful, but the possibility of such a combination should be considered in attempting to explain the origin of human solitary bone cyst.—*Arch. Path.*, 32:200-210, August 1941.

J. Edward Gilda.

Osteopetrosis: Four Cases in One Family

By Norman L. Higinbotham and Stuart F. Alexander

The author presents four cases of osteopetrosis, otherwise known as "marble bones," or Alberg-Schönberg disease, found in one family. All four cases present marked mandibular prognathism. The author states: "Infections of the bone offer the most serious hazard to these patients. In particular, infections of the jaw are to be dreaded, as they are the most dangerous to the life of the patient. With changes in the jaw bones, there is a malocclusion and spreading of the teeth as well as the basic decrease in the blood supply of the bone. These factors favor the development of dental infections, which spread into the maxilla or mandible. The resulting osteomyelitis is most resistant and frequently kills the patient." There is marked sclerosis of the cancellous and cortical bone. The bones may or may not be fragile. There is a tendency toward obliteration of the marrow cavity, with the possibility of developing anemia. The roentgenogram findings presented with these cases show marked sclerosis with almost complete loss of cancellous structure. All blood chemistry studies have failed to afford any characteristic findings. The etiology is unknown.—*Am. J. Surg., N. S.*, 53:444, September 1941.

N. S. Simmons.

Practical Control of Dental Caries

By Joan K. Savage

The practical control of dental caries does not necessarily involve the discovery of the etiology, nor the carrying out of more numerous operative procedures. The first step should aim at the education of the general public to the value of oral hygiene, the necessity of an adequate well-balanced diet and the desirability of routine dental examination.

Furthermore, the most vulnerable point of attack is the education and treatment of the child. Preventive work in the child can exclude much loss of tooth structure later in life. Adequate control of this disease involves not only the oral health, but also the general health of the community.—*D. J. Australia*, 13:295, July 1941.

A. Pearson.

Fusospirochetal Disease of the Mouth and Its Complications

By E. Lloyd Wilbur

Fusospirochetal disease of the mouth is discussed from the standpoint of etiology, predisposing factors, pathology, diagnosis, complications and treatment.

A definite etiology for this disease has not been established yet. However, the spirochetes and the fusiform bacilli in constant association must be considered as probably the causative agents. The chief predisposing conditions are blood dyscrasia and vitamin deficiency.

The basic lesion is characterized by necrosis and ulceration. From the standpoint of diagnosis, the disease is relatively easy to recognize. Soreness is present quite early. Later, inflammatory changes with ulceration occur. The odor itself is almost diagnostic, and, in conjunction with a smear, will assure a diagnosis in many cases.

Complications may occur either by direct extension or by way of the blood stream, usually the venous channels.

In mild cases in children, the treatment may consist of local applications or nothing at all. In more extensive cases, local measures together with some systemic treatment (arsenicals) are usually most effective.—*D. J. Australia,* 13:320, July 1941.

A. PEARSON.

Studies on Masticatory Efficiency: Part 3: Muscle Strength of Normal and Fluorosed Rats

By REIDAR F. SOGNNAES

IT has been suggested by many investigators that the mechanical force of mastication is an important factor in rat caries produced by coarsely ground diets. Incorporation of fluorine in these diets is known to reduce the caries lesions. Such a condition might result from the action of fluorine on the muscle function. This possibility was examined by measurements of the muscle strength of fluorosed and normal rats. No remarkable difference was found. The results seem to exclude the possibility that the inhibitory effect of fluorine in rat caries is due to any decrease in muscle strength and masticatory force.—*Am. J. Orthodontics,* 27: 458, August 1941.

FINN BRUDEVOLD.

Chronic Hyperplasia or Focal Osteitis Fibrosa of the Maxilla

By E. D. D. DAVIS

THE writer believes focal osteitis leontiasis ossea and osteitis deformans to be forms of one disease, caused neither by dental or nasal sinus sepsis nor traumatism. The abnormal bone is more susceptible to osteomyelitis, necrosis and local secondary sepsis than normal bone. The author discusses the clinical and laboratory findings associated with the disease and compares the lesions directly with other lesions involving the maxilla, such as osteomas, fibromas and cysts. The process is characterized by a slow painless enlargement of the alveolar process in the molar region of the maxilla. Passing forward, it obliterates the canine fossa and the whole surface of the maxilla is thickened, without signs of inflammation. The enlargement is confined to the outer surface of the alveolar process and its inner surface shows no swelling, in distinction from the picture that a tumor or a cyst might present.—*J. Laryng. & Otol.,* 56:179, May 1941.

N. S. SIMMONS.

Calcium and Phosphorus Studies in Normal People

By J. DOUGLAS ROBERTSON

A STUDY was made of serum calcium, serum inorganic phosphorus and serum phosphatase in sixty normal persons between the ages of 20 and 40, and in fifteen healthy persons aged 60 to 78. Serum calcium and inorganic phosphorus were not found to be significantly different in the two groups. The serum phosphatase was slightly raised in the older group.

The influence of a low calcium intake in depressing the calcium excretion was investigated in nine normal subjects. The results were compared with 139 similar observations from other workers. With a lower calcium intake, the daily output was restricted. The approximate equilibrium between intake and output was reached when the daily intake of calcium was 0.44-0.45 gm. for a 70-kg. subject.—*Lancet,* 2:97, July 26, 1941.

FINN BRUDEVOLD.

Behandling av taender med gangraenost sonderfallen pupa (Treatment of Teeth with Gangrenous Pulp)

By CARL PRUTZ

THE results of more than fifteen years' experience in treating gangrenous teeth with the Callahan-Johnston method is presented. The root canal is cleaned out and sterilized with 30-50 per cent sulfuric acid, and subsequently neutralized with a saturated solu-

tion of sodium bicarbonate. Before the filling of the root canal is undertaken, the canal is dehydrolized with absolute alcohol (98.4 per cent). Any excess of alcohol is removed with cotton and the canal is filled with resin-chloroform, 7 parts to 100. Chloropercha is then placed in the canal, which is finally plugged with a guttapercha point.

Two hundred fifty cases have been controlled roentgenographically and clinically for at least three years. Sixty-five per cent of the treated teeth had one canal, 20 per cent two canals and 15 per cent three or more canals. Complete regeneration in the apical region occurred in 74.4 per cent and incomplete regeneration in 15.2 per cent; while no changes occurred in 10.4 per cent. Of the twenty-six teeth in the last group, ten were upper cuspids.—*Svensk Tandl. Tidskr.*, 33:193, 1941.

FINN BRUDEVOLD.

Does Salivary Calcium and Phosphorus Composition Differ Significantly in Caries-Free and Caries-Active Individuals?

By HERMANN BECKS and W. W. WAINWRIGHT

SALIVARY analyses of ninety carefully selected, caries-free persons between the ages of 11 and 53 years and 108 caries-active between the ages of 10 and 58 years revealed no significant differences between the groups on the basis of rate of flow and calcium and phosphorus content. It was found that extremes of age and rate of flow influenced the calcium and phosphorus averages, regardless of the presence or absence of dental caries. In this extensive study, the authors conclude that the reported differences in calcium and phosphorus content of the saliva between caries-free and caries-active groups, on resting saliva, must be attributed to the variation in the selection of material, and their findings emphasize the lack of any relationship between salivary rate of flow and calcium and phosphorus content. —*J. D. Res.*, 20:171-188, June 1941.

JAMES T. GINN.

Action of Actinomycetes-Like Organism from Root Canals

By MARY C. CROWLEY

THIS is a study of an actinomycetes-like organism found in root canals. Seven strains of this organism were isolated in a beef infusion broth pH 7.4. These strains presented similar characteristics in that they were isolated with some difficulty, growing under partial anaerobic conditions and only in media enriched with body fluids. Smears made from colonies and broth cultures showed non-acid fast, gram-positive fine branching filaments. They formed branched mycelia with structures similar to the true *Actinomycetes bovis*. This organism is saprophytic, intraperitoneal animal inoculation failing to produce any lesion.—*J. D. Res.*, 20:189-194, June 1941.

JAMES T. GINN.

Significance of Salivary Analyses upon Small Groups of Subjects

By M. L. DEAKINS, V. D. CHEYNE, B. G. BIBBY and M. VANKESTEREN

EIGHTEEN different chemical and bacteriologic studies were made on twenty specimens of stimulated whole saliva taken from different subjects. It was found that no definite relation existed between any constituent studied and the rate of flow, either on the amount per cubic centimeter basis or on an amount per cubic centimeter per minute basis. When the samples were divided into "fast" and "slow" groups of eight cases each, eliminating four cases nearest the mean for the group of twenty, there was a significant difference between the rates of flow of total nitrogen and protein nitrogen, acid-neutralizing power, base-neutralizing power, total buffer capacity, total solids, total bacteria and volume. The authors conclude that this study emphasizes the difficulty of interpreting the results of salivary analysis in small groups, no matter how they are calculated.—*J. D. Res.*, 20:161-170, June 1941.

JAMES T. GINN.

Successful Treatment of Noma with Solution of Formaldehyde

By SIM I. MCMILLEN

SIX cases of noma successfully treated with a solution of formaldehyde are reported in children between the ages of 1 year and 2 years. The disease is characterized by a foul, gangrenous, rapidly spreading ulcer with extensive destruction of tissue in and about the oral cavity, and early death. An

undiluted solution of formaldehyde was applied to the ulcer daily, the treatment proving successful in seven consecutive cases, which is rather remarkable, since the mortality rate is about 90 per cent.

Since *S. vincenti* and *B. fusiformis* are the predominating organisms in tropical ulcer, noma, Vincent's infection and sloughing phagedena, and formaldehyde has been used successfully in the treatment of tropical ulcer and noma, the author suggests that formaldehyde might be of some value in treating certain cases of Vincent's infection and hospital (sloughing) phagedena.—*Am. J. Dis. Child.*, 62:590-595, September 1941.

JAMES T. GINN.

Radiosensitivity of Adamantinoma

By H. T. KIMM

IN a review of the literature, the author concludes that the current theory that the adamantinoma is radioresistant is not substantiated by these reports, nor is it borne out by his own clinical experience with this tumor. In nine cases of this tumor treated with roentgen rays or radon seeds, the tissues secured before and at varying periods after irradiation were compared and it was found that intensive interstitial and moderately high roentgen dosage caused marked regressive changes in these tumors, although the typical differentiated elements were not materially affected. In one case of the maxilla of the basal adenoid cystic type, almost complete destruction of the tumor was observed about one and one-half months after irradiation. Intensive irradiation resulted in slight clinical regression of the tumors in two cases. The author concludes that differentiated tumors of this type are highly radioresistant, while undifferentiated tumors, such as the basal cell type, are comparatively radiosensitive.—*Chinese M. J.*, 59:497-507, June 1941.

JAMES T. GINN.

Herpes Labialis After Sulfapyridine and T.A.B. Therapy

By C. E. VAN ROOYNEN, A. J. RHODES and A. C. EEING

THE authors review the subject of herpes, pointing out that all of the various herpetic outbreaks in man (except herpes zoster), which are known under a variety of names, have one common virus. They also point out that aphthous ulcers in the mouth are caused by the herpes virus which is commonly found in the saliva.

Twenty-seven patients developed herpes after a course of sulfapyridine (25 gm. in four days) followed by typhoid-paratyphoid vaccine shock. As the vaccine or sulfapyridine alone did not have the effect, it is concluded that the sulfapyridine enhances the herpetogenic effect of protein shock. Additional evidence demonstrates the occurrence of herpes virus in saliva, especially in the winter months.—*Brit. M. J.*, 2:298, August 21, 1941.

BASIL G. BIBBY.

Influence of the Formative Dental Organs on the Growth of the Bones of the Face

By L. W. BAKER

IN experiments conducted on young rats, cats and pigs, various tooth germ crypts were aspirated unilaterally. An arrest of the development of the facial bones on the side where and in the direction in which the removed teeth would normally have grown could be demonstrated in each case. Hence, the erupting teeth play an important rôle in the growth and normal building of the facial skeleton, and influence profoundly the structure of the cranial base. Any condition retarding the developing dentition, such as deficiency disease, endocrine disturbance or serious childhood disease, may be considered a force tending toward deformity of the face and jaws.—*Am. J. Orthodontics*, 27:489, September 1941.

H. B. MCCAULEY.

Acute Infectious Gingivostomatitis

By T. F. MCNAIR SCOTT, A. J. STEIGMAN and J. H. CONVEY

METHODS for the isolation of the herpes simplex virus in the saliva, for the determination of the development of antibodies in the blood and for bacteriologic studies, were employed in twenty-one cases of "acute infectious gingivostomatitis." Its etiology is clarified by the isolation of the virus of herpes simplex from the mouths of the patients and by the development of herpes neutralizing antibodies in their blood during convalescence. The clinical picture of an acute systemic infection characterized by fever, irritability, soreness of the mouth, red-

ness and swelling of the gums, oral fetor and regional lymphadenopathy is indicative of primary herpetic stomatitis. The descriptive terms "catarrhal," "aphthous," "ulcerative," and "membranous," employed in the usual classification of stomatitis, apply to different degrees of severity of the herpetic lesion. It is suggested that the term "herpetic stomatitis" be applied to this common gingivostomatitis. The primary disease is the stomatitis of young children, which is usually accompanied by constitutional symptoms which do not always parallel in severity the oral lesions. The recurrent disease may occur at any age, after a primary infection with herpes, and is analogous to recurrent herpes of the lips and elsewhere. The local lesion may be severe, but, as a rule, the constitutional symptoms are mild, if present at all.

Infection spreads readily through a susceptible population. The evidence suggests possible sources of endemic foci with special reference to mothers.—*J.A.M.A.*, 117:999-1005, September 20, 1941.

J. EDWARD GILDA.

Cause and Control of Shrinkage Porosity in Inlay Casting

By JOHN L. WILSON

FUNDAMENTAL principles of metallurgy involved in casting are discussed, with explanations of alloys, dendritic structure and cooling curves. Dendrites begin their formation in the upper temperatures of the cooling range and continue growing as the temperature is lowered. If molten metal is not available to complete the formation of crystals, voids are formed and shrinkage porosity is produced.

Photomicrographs illustrate porous and non-porous castings. Suggestions for supplying molten metal to the areas freezing last are given. The simplest method is the use of a sprue about 5 mm. in length and of 12 or 14 gage wire. The surface tension of the gold prevents any molten gold from falling into the sprue hole. If a medium size sprue is used, a reservoir should be made. Precaution should be taken to use a large reservoir; for, if it is too small, metal will be drawn from the casting and thus a porous condition be produced around the base of the sprue.—*D. Digest*, 47:9, September 1941.

RALPH W. PHILLIPS.

Rontgenologische Kontrolle pulpachirurgisch behandelter Milchzähne (X-Ray Control of Deciduous Teeth with Root-Canal Treatment)

By BIRGER OSTMAN and HAROLD THYBERG

THIS investigation covers x-ray findings on the surrounding tissues after partial pulpotomy of 1,055 deciduous molars. Only teeth with vital, intact or superficially inflamed pulps were subject to treatment. In most cases, the pulp was devitalized and the drugs employed were chloramin, 30 per cent hydrogen peroxide and eugenol. The compounds used as sealing paste consisted of liquid of olein plus zinc oxide and xeroform or eugenol plus zinc oxide and xeroform.

Of the 1,055 teeth that were studied, 979, or 92.3 per cent, gave no evidence of periapical inflammation at the beginning of treatment. In this group, 54.4 per cent remained satisfactory from a roentgenologic point of view during the period of investigation, while 42.4 per cent were considered unsuccessful. A small group, 3.2 per cent, showed inflammation at some intermediary x-ray check-up, but gave evidence of healing at a later date. Seventy-six of the 1,055 teeth showed pathologic changes in the pericementum at the beginning of the treatment. Forty of these, or 52.6 per cent, did not show any improvement, 14.5 per cent did, and in 32.9 per cent healing was complete. A group of 297 of the treated teeth, or 28.2 per cent, revealed resorption processes in the root canal, indicating chronic inflammation of the remaining pulp.—*Acta Odontol. Scandinav.*, 3:3, June 1941.

FINN BRUDEVOLD.

Comparison of Dental Caries on Buccal and Proximal Surfaces of Premolar Teeth

By J. M. DUNNING

HYATT'S figures on 12,645 employes of a large insurance company covering the entire age range from 17 to 60 years, and a wider range in economic and in social status, were used. The measure of incidence of caries per hundred tooth surfaces was based entirely on the teeth actually present in the mouth, as regards both the number of lesions found and the number of tooth surfaces present at the time of the examination. In

the groups, proximal premolar caries occurred almost entirely before the age of 34, and buccal, hence by inference cervical, premolar caries steadily throughout the entire age range of the study.—*J. D. Res.*, 20:195-201, June 1941.

J. EDWARD GILDA.

Some Factors in Secretion by Submaxillary Glands of Cats

By J. H. WILLS

IN a study of the effects of chorda tympani stimulation upon the secretory response of the submaxillary glands of cats, it was found that a stimulation frequency of nine shocks per second from a thyratron resulted in a higher initial salivary output and better maintenance of output rate than when any other frequency was employed. Continued stimulation of the gland gave changes in the rate of secretion analogous to muscle tension changes under like conditions. Injection of pilocarpine caused a greater flow of saliva for a given blood flow than did chorda stimulation. This may explain the loss of potassium from the gland observed during secretion after pilocarpine administration. At rates below 0.03 cc. per minute, the potassium concentration of the saliva was rapidly increased.—*Am. J. Physiol.*, 134:441, October 1941.

H. B. MCCAULEY.

Unspezifische Allgemeinsymptome bei chronischer dentaler Infektion (Non-Specific Systemic Symptoms Associated with Chronic Dental Infection)

By SOPHUS BUGGE

CHRONIC apical paradentitis may have a distinctly deleterious effect upon the whole organism and affect the mechanism of vegetative regulation as well as the new formation of blood. Several hundred patients suffering from chronic apical paradentitis were examined as to temperature, pulse rate, sedimentation rate, total leukocyte count, Schilling's differential blood counts, hemoglobin and body weight. Thirty cases are presented to demonstrate certain pathologic changes which are reduced or disappear after extraction of the infected teeth. There seems to be an individual combination of positive symptoms in every patient, differing with respect to the quality and strength of the pathologic reactions. There may be a reactive increase of symptoms in connection with defocalization. This is most markedly shown by changes in temperature, total leukocyte counts, Schilling's differential blood counts and sedimentation rate.—*Acta Odontol. Scandinav.*, 3:33, June 1941.

FINN BRUDEVOLD.

Rickets

By MEYER EGGNATZ

EVIDENCE of rickets in infants may be observed clinically before the fourth or fifth month after the onset of the disease. Important signs are enlargement of the head, beading of the ribs, shrinking and distortion of the thorax, enlargement of the abdomen, swelling of the wrists and ankles and curving of the long bones. Eruption and development of the teeth are usually retarded. The deciduous teeth may be normal, but the permanent teeth often exhibit enamel of poor quality, predisposed to caries. Teeth affected are small, comparatively soft, discolored and easily broken. The effects are consistently found in teeth which are in the process of formation during the period of the disease. There is no conclusive evidence that rickets is a factor in dental decay. Field and laboratory studies indicate a possible relationship between rickets and hypoplasia of enamel, and between rickets and malocclusion, especially class I (Angle).—*Am. J. Orthodontics*, 27:379, July 1941.

H. B. MCCAULEY.

Actinomyces of the Human Mouth

By BASIL G. BIBBY and HOLMES T. KNIGHTON

INVESTIGATION on the occurrence of branched organisms from the mouth indicate that the present types of classification are inadequate. Examination of smears failed to reveal any branched organisms. Culturally, numerous branched and clubbed aerobic organisms were demonstrated. Occasionally, similar types were found anaerobically. No attempt was made to classify these forms, but the aerobic organisms showed definite morphologic similarity to *A. bovis*.—*J. Infect. Dis.*, 69:148, September-October 1941.

A. PEARSON.

ANNOUNCEMENTS

ANNOUNCEMENTS must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

CALENDAR OF MEETINGS

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF ORAL DIAGNOSIS, New York, N. Y., November 13-14.

AMERICAN ASSOCIATION OF ORTHODONTISTS, New Orleans, La., March 16-19, 1942.

CHICAGO DENTAL SOCIETY MIDWINTER MEETING, February 23-26, 1942.

DALLAS MIDWINTER DENTAL CLINIC, Dallas, Texas, January 26-28, 1942.

DISTRICT OF COLUMBIA DENTAL SOCIETY, second and fourth Tuesdays in each month from October to June at the United States Public Health Service Auditorium, Washington.

DENTAL PROTECTIVE ASSOCIATION, Chicago, Ill., December 15.

DENVER DENTAL ASSOCIATION, Denver, Colo., January 25-28, 1942.

FIVE STATE POST GRADUATE CLINIC, Washington, D. C., March 8-12, 1942.

GREAT LAKES SOCIETY OF ORTHODONTISTS, Ann Arbor, Mich., November 3-4.

MIDCONTINENT DENTAL CONGRESS, St. Louis, Mo., November 17-19.

NATIONAL BOARD OF DENTAL EXAMINERS, December 1-2.

UNIVERSITY OF NEBRASKA DENTAL ALUMNI ASSOCIATION, Lincoln, November 14-15.

GREATER NEW YORK DENTAL MEETING, New York, December 1-5.

NORTH ATLANTIC ORTHODONTIC SOCIETY, New York, N. Y., January 21, 1942.

ODONTOLOGICAL SOCIETY OF WESTERN PENNSYLVANIA, Pittsburgh, November 11-13.

PAN AMERICAN ODONTOLOGICAL ASSOCIATION, New York, N. Y., December 3.

GREATER PHILADELPHIA ANNUAL MEETING, February 3-6, 1942.

STATE SOCIETIES

November

Ohio, at Columbus (10-12)

December

Florida, at Hollywood (8-10)

March

Minnesota, at St. Paul (3-5)

STATE BOARDS OF DENTAL EXAMINERS

California, at San Francisco, December 15. Kenneth I. Nesbitt, 515 Van Ness Ave., San Francisco, Secretary.

Connecticut, at Hartford, November 25-29. Clarence C. Brooks, New London, Recorder.

Iowa, at Iowa City, December 15-19. Harry G. Bolks, Sioux City, Secretary.

Minnesota, at Minneapolis, December 15-20. Elmer J. Sundby, 805 Medical Arts Bldg., Duluth, Secretary.

New Jersey, December 8-13. Walter A. Wilson, 150 E. State St., Trenton, Secretary.

Pennsylvania, at Philadelphia and Pittsburgh, December 9-13. Reuben E. V. Miller, 61 N. 3rd St., Easton, Secretary.

Texas, at Houston, December 8-11. B. Carl Holder, 1009 Nixon Bldg., Corpus Christi, Secretary.

Wisconsin, at Milwaukee, December 15-19. S. F. Donovan, Tomah, Secretary.

AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF ORAL DIAGNOSIS

THE eighth annual congress of the American Association for the Advancement of Oral Diagnosis will be held in New York, N. Y., November 13-14, at the New York Academy of Medicine. The papers, discussions and exhibits will cover oral diagnosis with special attention to the basic sciences. Members of the medical and the dental professions in the United States and countries of the Western Hemisphere are invited to attend and can obtain programs by communicating with the secretary.

H. JUSTIN ROSS, *Secretary*,
515 Madison Ave.,
New York, N. Y.

AMERICAN ASSOCIATION OF ORTHODONTISTS

THE 1942 meeting of the American Association of Orthodontists will be held at the Roosevelt Hotel, New Orleans, La., March 16-19, 1942. This will be a pan American congress. An invitation is extended to orthodontists in Central and South America.

MAX E. ERNST, *Secretary,*
1250 Lowry Medical Arts Bldg.,
St. Paul, Minn.

CHICAGO DENTAL SOCIETY MIDWINTER MEETING

THE 1942 Midwinter Meeting of the Chicago Dental Society will be held at the Palmer House, February 23-26.

GLENN E. CARTWRIGHT, *President.*

DALLAS MIDWINTER DENTAL CLINIC

THE Dallas Midwinter Clinic, sponsored by the Dallas County Dental Society, will hold its fifteenth annual meeting in the Hotel Adolphus, Dallas, Texas, January 26-28, 1942. The following clincians will present a three-day study course: Sanford M. Moose, San Francisco, Calif.; subject: "Oral Surgery and Diagnosis." Ernest R. Granger, Mt. Vernon, N. Y.; subject: "Operative Dentistry." R. O. Schlosser, Chicago, Ill.; subject: "Denture Prosthesis." Members of the Dallas County Dental Society will present table clinics. The fee for the full three-day course, including the dinner, is $15. All members of the American Dental Association are invited. 1942 membership cards should be presented on registration.

PHELPS J. MURPHEY, *Chairman,*
Publicity Committee,
Fairmount and Welborn Sts.,
Dallas, Texas.

DENTAL PROTECTIVE ASSOCIATION

THE annual meeting of the Dental Protective Association of the United States will be held at the Palmer House, Chicago, December 15, at 4 p.m. All members are urged to be present.

E. W. ELIOT, *Secretary,*
55 E. Washington St.,
Chicago, Ill.

DENVER DENTAL ASSOCIATION

THE next meeting of the Denver Dental Association will be held at the Shirley Savoy Hotel, Denver, January 25-28, 1942.

ALBERT P. HORTON, *Director,*
206 Metropolitan Bldg.,
Denver, Colo.

FIVE STATE POST GRADUATE CLINIC

THE annual Five State Post Graduate Clinic of the District of Columbia Dental Society will be held at the Mayflower Hotel, Washington, D. C., March 8-12, 1942.

WILLIAM D. CURTIS, *Chairman,*
Publicity Committee.

GREAT LAKES SOCIETY OF ORTHODONTISTS

THE Great Lakes Society of Orthodontists will meet November 3-4 at Ann Arbor, Mich.

RICHARD E. BARNES, *Secretary,*
1704 Republic Bldg.,
Cleveland, Ohio.

MIDCONTINENT DENTAL CONGRESS

THE Midcontinent Dental Congress will be held in St. Louis, Mo., November 17-19.

VAL H. FREDERICH, *Chairman,*
Exhibit Committee,
Arcade Bldg.,
St. Louis, Mo.

NATIONAL BOARD OF DENTAL EXAMINERS

THE National Board of Dental Examiners will hold its next session for the examination of candidates in Parts I and II, December 1-2, in schools where there are five or more candidates. For further information and application blanks, address

MORTON J. LOEB, *Secretary,*
66 Trumbull St.,
New Haven, Conn.

UNIVERSITY OF NEBRASKA DENTAL ALUMNI ASSOCIATION

THE University of Nebraska Dental Alumni Association will hold its annual homecoming meeting November 14-15 at the college, with various lectures, demonstrations and clinics by the faculty and guest speakers. Pittsburgh vs. Nebraska football game Saturday afternoon.

RALPH L. IRELAND, *Secretary,*
College of Dentistry,
Lincoln.

GREATER NEW YORK DENTAL MEETING

THE Greater New York Dental Meeting

will be held December 1-5 at the Hotel Pennsylvania, New York.

WALTER A. QUINN, *Chairman,*
Press & Publication,
Room 106A,
Hotel Pennsylvania,
New York.

NORTH ATLANTIC ORTHODONTIC SOCIETY

THE fourteenth semi-annual meeting of the North Atlantic Orthodontic Society will be held January 21, 1942, in the Hotel Pennsylvania, New York, N. Y. Members of the dental and medical professions are invited.

A. G. KRAMER, *Secretary,*
1111 E. 15th St.,
Brooklyn, N. Y.

ODONTOLOGICAL SOCIETY OF WESTERN PENNSYLVANIA

THE annual meeting of the Odontological Society of Western Pennsylvania will be held November 11-13 at the William Penn Hotel, Pittsburgh.

W. EARLE CRAIG, *Secretary,*
206 Jenkins Bldg.,
Pittsburgh.

PAN AMERICAN ODONTOLOGICAL ASSOCIATION

THE next annual dinner meeting of the Pan American Odontological Association will be held December 3 at 7 p.m., at the Hotel Pennsylvania, New York, N. Y.

RALPH HOWARD BRODSKY,
Executive Secretary,
14 E. 81st St.,
New York, N. Y.

GREATER PHILADELPHIA ANNUAL MEETING

THE Greater Philadelphia Annual Meeting, which will be a combined meeting of the Pennsylvania State Dental Society and the Philadelphia County Dental Society, will be held February 3-6 at the Benjamin Franklin Hotel.

ABRAM COHEN, *Chairman,*
Publicity Committee,
269 S. 19th St.,
Philadelphia, Pa.

FLORIDA STATE DENTAL SOCIETY

THE fifty-eighth meeting of the Florida State Dental Society will be held at the Hollywood Hotel, Hollywood, December 8-10.

WILLIAM P. WOOD, JR., *Secretary,*
442 W. Lafayette St.,
Tampa.

MINNESOTA STATE DENTAL ASSOCIATION

THE fifty-ninth annual session of the Minnesota State Dental Association will be held in the Municipal Auditorium, St. Paul, March 3-5, 1942.

L. M. CRUTTENDEN, *Secretary,*
498 Lowry Medical Arts Bldg.,
St. Paul.

OHIO STATE DENTAL SOCIETY

THE seventy-sixth annual meeting of the Ohio State Dental Society will be held at the Neil House, Columbus, November 10-12. All A.D.A. members are invited.

EDWARD C. MILLS, *Secretary,*
255 E. Broad St.,
Columbus.

BOARD OF DENTAL EXAMINERS OF CALIFORNIA

THE next examination to be conducted by the Board of Dental Examiners of California for a license to practice dentistry in California will be held in San Francisco commencing December 15 at the Physicians and Surgeons College of Dentistry for the theory portion of the examination and at the University of California, College of Dentistry for the practical portion of the examination. All credentials must be in the office of the Secretary at least twenty days prior to the date of the examination. Address all communications to

KENNETH I. NESBITT, *Secretary,*
515 Van Ness Ave.,
San Francisco.

THE CONNECTICUT DENTAL COMMISSION

THE Connecticut Dental Commission will meet in Hartford, November 25-29, for the examination of applicants for license to practice dentistry and dental hygiene and to transact any other business proper to come before it. Applications should be in the hands of the recorder at least ten days before the meeting. For application blanks and further information, apply to

CLARENCE G. BROOKS, *Recorder,*
New London.

IOWA STATE BOARD OF EXAMINERS

THE Iowa State Board of Examiners will hold its next examination in Iowa City, December 15-19, for those desiring a license as dentist or as dental hygienist. Address Harry G. Bolks, secretary of the board, Sioux City, for instructions regarding registration for examination, etc.

MINNESOTA STATE BOARD OF DENTAL EXAMINERS

THE Minnesota State Board of Dental Examiners will hold its next examination at the School of Dentistry, University of Minnesota, Minneapolis, December 15-20. For information and application, address

ELMER J. SUNDBY, *Secretary,*
805 Medical Arts Bldg.,
Duluth.

STATE BOARD OF REGISTRATION AND EXAMINATION IN DENTISTRY OF NEW JERSEY

THE State Board of Registration and Examination in Dentistry of New Jersey will hold its annual examinations commencing December 8 and continuing for five days thereafter. On application to the secretary, a copy of the requirements and rules, instruction sheet and preliminary application will be forwarded. Any person desiring to become a candidate must file the preliminary application blank, together with the examination fee of $25 on or before September 1.

WALTER A. WILSON, *Secretary,*
150 E. State St.,
Trenton.

DENTAL COUNCIL AND EXAMINING BOARD OF THE COMMONWEALTH OF PENNSYLVANIA

THE Dental Council and Examining Board of the Commonwealth of Pennsylvania will conduct examinations in Philadelphia and Pittsburgh, December 9-13. For information and application blanks, address the Department of Public Instruction, Bureau of Professional Licensing, Dental Division, Harrisburg.

REUBEN E. V. MILLER, *Secretary,*
61 N. Third St.,
Easton, Pa.

TEXAS BOARD OF EXAMINERS

THE Texas Board of Dental Examiners will hold its next examination December 8-11 in Houston. Application must be filed at least ten days prior to date of examination. For further information, address

B. CARL HOLDER, *Secretary,*
1009 Nixon Bldg.,
Corpus Christi.

STATE OF WISCONSIN BOARD OF DENTAL EXAMINERS

THE next examination will be held December 15-19 at Marquette University Dental School, Milwaukee. Applications and information may be had by writing to the secretary. Applications and required fee must be in the secretary's office by December 5.

S. F. DONOVAN, *Secretary,*
Tomah.

SOCIETY OF THE NEW YORK HOSPITAL LEWIS CASS LEDYARD, JR., FELLOWSHIP

THE Lewis Cass Ledyard, Jr., Fellowship was established in 1939 by a gift from Mrs. Lewis Cass Ledyard, Jr., in memory of her husband, a governor of the New York Hospital. The income, amounting to approximately $4,000 annually, will be awarded to an investigator in the fields of medicine and surgery, or in any closely related field. The recipient will be required to submit reports of his work under the fellowship, either at stated intervals or at the end of the academic year. The research work is to be carried on at the New York Hospital and Cornell Medical College. The fellowship will be available July 1 at the beginning of the academic year. Applications for the year 1942-1943 should be in the hands of the committee by December 15. It is expected that the award will be made by March 15, 1942. Application for this fellowship should be addressed to the Committee of the Lewis Cass Ledyard, Jr. Fellowship, the Society of the New York Hospital, 525 E. 68th St., New York, N. Y.

DENTAL SUPPLIES FOR GREAT BRITAIN AND CHINA

THE United States Section of the International College of Dentists is sponsoring a movement to aid the dentists of Great Britain and China by providing them with badly needed supplies, instruments and small equipment used in the practice of dentistry. These will be collected and sent abroad by the Medical and Surgical Relief Committee

of America. Any member of the dental profession or dental trade who can aid in this movement by contributions of such material is asked to communicate with Mrs. Rodgers Balcom, executive chairman of the Medical and Surgical Relief Committee of America, 420 Lexington Ave., New York, N. Y.

ELMER S. BEST, *Registrar,*
Medical Arts Bldg,
Minneapolis, Minn.

BRONX HOSPITAL VOLUNTEERSHIPS

THE Dental Department of the Bronx Hospital offers a number of volunteerships to recent graduates in dentistry in the following branches: oral surgery and anesthesia; surgical pyorrhea; orthodontia; root canal therapy, and operative dentistry. Those offering satisfactory credentials indicating their interest in the clinical study of dental surgery as a health problem will receive consideration. Address applications to the Dental Department, Bronx Hospital, Fulton Ave. at 169th St., Bronx, New York. Appointments will begin in July.

DAVID WURZEL, *Director,*
Dental Department.

CARNEGIE FELLOWSHIPS FOR RESEARCH AND GRADUATE WORK IN THE DENTAL SCIENCES

AIDED by the Carnegie Corporation, the School of Medicine and Dentistry of the University of Rochester offers scholarships paying $1,200-$1,800 per annum to well-trained dental graduates who are interested in research or who desire special training in the basic medical sciences as an aid to dental teaching or practice. A few fellowships also offer part-time training in oral surgery. On appointment, the applicant is given the title of Carnegie Dental Fellow and, according to his interest, works as a graduate student in one of the departments of the medical schools. Here, excellent laboratory facilities, adequate direction and close contact with various investigations related to medicine and dentistry help his research and study. Fellows are encouraged to attend medical classes, seminars and staff meetings and, when opportunity presents itself, to participate in class instruction and scientific meetings. Contact with the problems of clinical dentistry may be maintained through the Oral Surgery Clinic of the Strong Memorial Hospital and the Rochester Dental Dispensary. With the approval of the Graduate Council of the university, Fellows may prepare themselves for the degree of M.S. or Ph.D. Application should be made to

GEORGE H. WHIPPLE, *Dean,*
School of Medicine and Dentistry,
Rochester, N. Y.

POSTGRADUATE COURSE IN PRACTICE MANAGEMENT, SCHOOL OF DENTAL AND ORAL SURGERY, COLUMBIA UNIVERSITY

THE School of Dental and Oral Surgery, Columbia University, offers a course in practice management for qualified graduates in dentistry under the direction of George Wood Clapp, D.D.S., October 7, 9, 14 and 16 and November 6, 13, 18 and 25. Admission to the course is limited to members of the American Dental Association. For further information, address the assistant to the registrar, School of Dental and Oral Surgery, 630 W. 168th St., New York, N. Y.

COLUMBIA UNIVERSITY SCHOOL OF DENTAL AND ORAL SURGERY REFRESHER COURSES

COLUMBIA University School of Dental and Oral Surgery offers two short courses in periodontia for qualified graduates in dentistry January 23 to May 22, 1942, and September 18, 1942, to January 15, 1943.

A ten-day intensive course will include lectures, discussions, demonstrations and clinical practice. An extended course, one half day per week for two sesions, will included lectures, discussions and supervised technical treatment. Both courses will be conducted by Frank E. Beube, Isador Hirschfeld, Harold J. Leonard (executive officer) and Arthur H. Merritt.

The courses given at the School of Dental and Oral Surgery, Columbia University, are designed to be "refresher" courses; i.e., to bring practitioners abreast of modern undergraduate teaching in theory and practice in the subject. Admission to the course is limited to qualified graduates in dentistry who are members in good standing of the American Dental Association or recognized sectional organizations. In the case of foreign applicants, membership in the applicants' national dental organization is required.

Information can be obtained from the registrar, School of Dental and Oral Surgery, 630 West 168th Street, New York, N. Y.

UNITED STATES PATENTS APPLICABLE OR PERTAINING TO DENTISTRY ISSUED DURING AUGUST AND SEPTEMBER

August 5

No. 2,251,501, ARTHUR E. SMITH. Syringe ampoule.

No. 2,251,527, ARTHUR E. Smith. Syringe and ampoule.

No. 2,251,749, HERBERT MANDUS. Dental appliance.

No. 2,251,853, ABRAHAM JOTHY PANDIYAN. Toothbrush.

No. D-128,681, LUIS L. REINOLD. Design for a toothbrush.

No. D-128,685, EDWARD L. CHOTT. Design for a dental brush.

August 12

No. 2,251,986, KARL CONNELL. Apparatus and method for the administration of gases.

No. 2,251,996, SAMUEL D. GOLBERG. Process for making anesthetic bases and intermediates.

No. 2,252,251, FREDERICK BREMNER COOPER. Rotary tooth cleaner.

No. 2,252,530, RAYMOND LLEWELLYN SWEENY and GEORGE ALBERT RICHARDS. X-ray timing apparatus.

No. 2,252,575, EARL E. LONGFELLOW. Surgical tool for cutting plaster casts.

August 19

No. 2,252,713, SAMUEL D. GOLDBERG. Local anesthetic intermediate and process for manufacturing the same.

No. 2,252,798, OLIN B. ARNOLD. Dental forceps.

No. 2,252,935, CARL EMIL LIEDBERG. Dentistry jaw.

No. 2,253,132, TOTTEN S. MALSON. Dental instrument.

No. 2,253,143, HARRY P. SIEGEL. Dental aspirator.

No. 2,253,210, NICHOLAS PSIHARIS. Gum massage device.

No. 2,253,222, JOSEPH BERTRAM. Pontic insert and dental bridge.

No. 2,253,259, RAYMOND WILSON. Die for x-ray mount presses.

August 26

No. 2,253,514, THOMAS W. FISHER. Fountain toothbrush.

No. 2,253,910, FRANZ LUENZ. Gum massage implement.

September 2

No. 2,254,365, EDWARD E. GRIFFITH. Convertible toothbrush.

No. 2,254,449, RICHARD E. RASMUSSEN. Injection syringe and protective sheath.

No. 2,254,556, JAY E. WAGNER. Anatomical dental flask.

No. 2,254,638, HEINER WIENAND. Method and apparatus for molding artificial teeth.

No. 2,254,854, HAROLD V. O'CONNELL. Anesthetic administering device.

No. D-129,322, WALTER S. CROWELL. Design for a mouth mirror.

September 9

No. 2,255,267, HUGH G. MORAN. Dental appliance.

No. 2,255,657, Hyman Freedman. Dental apparatus.

September 16

No. 2,256,344, Pyungtoo William Lee. Process of manufacturing nonshatterable vitreous denture bases.

September 23

No. 2,256,667, CLAIRE M. DORET. Tooth study model.

No. 2,257,050, EDWIN R. GOLDFIELD. Timing apparatus for x-ray exposures.

No. 2,257,069, JOSEPH DAYTON PEAK. Orthodontic bracket.

September 30

No. 2,257,141, ARTHUR L. WAUGH. Surgical knife.

No. 2,257,337, DONOVAN B. GRABLE. Dental amalgamator.

No. 2,257,669, WILLIAM H. BECKMANN. Artificial tooth and holder therefor.

No. 2,257,709, LOUIS P. ANDERSON. Dental appliance.